"NUCLEAR WEAPON CASUALTIES - EFFECTS ON PERSONNEL"

Final Report Contract AF33(600)-39504

Prepared For

Air Force Intelligence Center
Assistant Chief of Staff, Intelligence
Headquarters, United States Air Force
Washington 25, D.C.

1 May 1961

E. H. SMITH AND COMPANY, INC.

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SILVER SPRING, MARYLAND

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FORWARD

The purpose of this study was to develop data on selected medical effects for use as inputs to an over-all Nuclear Weapons Casualties Program sponsored by the Air Force Intelligence Center. The publication of this report does not constitute approval by the Air Force of the findings or conclusions herein.

SUMMARY

The purpose of this part of the report is to summarize the casualty data to be used. The summary will be given by Chapter and Section of the report.

1. Chapter I, Section 1.

The data to be used for early casualties from very high neutron plus gamma dose are given in Figure 1. This figure gives the average number of personnel who are capable of performing duties in the first eight hours after irradiation. For example, 50% of the personnel can perform duties if the irradiation level was 7000 r. However, the same 50% may not be included in the "effective" class for all times during the first eight hours. Thus, one group of 50% may be effective at one time and some of the group may be replaced by others at a later time. One cannot say, therefore, that the effective group will always consist of the same people.

2. Chapter I, Section 2.

The pertinent equation for calculation of effective dose at time t under an irradiation rate $\gamma(t)$ r/hour is given by 2.3 with $f = 0.20$ and $.75 \leq \epsilon^{-\beta} \leq .90$.

For partial body irradiation, the pertinent equation is 2.4 with $f = 0.41$.

3. Chapter I, Section 3.

It is concluded here that the RBE of γ or x-radiations relative to 250 kvp x-rays is unity if absorbed dose is used as calculated by Eq. 2.4, Section 2. It is further concluded that there is insufficient information to warrant setting the RBE of thermal or fast neutrons at a value other than unity relative to x or γ rays.

4. Chapter II, General Observations.

This Chapter contains some discussion of the effects of clothing and the effects of exposure to partial pulses from large weapons. Both of these subjects pertain more to the "environmental contract" than to this contract and, consequently, more discussion of these phenomena will appear in the final report on Contract AF33(600)-40617.

The pertinent data on thermal burn casualties are given in Table 8, which treats depth of burn for a given exposure; Table 2.1 which gives the time to hypovolemic shock versus depth of burn and area burned; and Section 4 which discusses clinical aspects of burn injury, incapacitation time, burns to critical areas, repair times and prognosis.

5. Chapter III, Section 1, Part 1.

The critical translational speed for injury is shown to be 20 ft/sec.

6. Chapter III, Section 1, Part 2.

It is shown that 5 psi overpressure should be taken as the critical overpressure for serious injury or death to personnel in rooms or buildings. 3 psi is shown to be reasonable for personnel in city streets.

7. Chapter III, Section 1, Part 3.

Figures 2, 3, 4 and 5 give the speeds obtained by men and missiles for the case of free translation in the open.

8. Chapter III, Section 2.

The critical overpressure for direct blast injury (i.e., injury from the shock wave alone with no translation) is shown to be 35 psi.

9. Chapter IV.

The only important class of combined injury is shown to be thermal-nuclear radiation injury. The pertinent conclusions are given at the very end of the Chapter.

I. CASUALTIES FROM NUCLEAR RADIATION

Section 1. Early Incapacitation from High Doses of γ-rays and Neutrons.

By far the most useful data on early incapacitation from γ-rays is contained in Reference 1. We wish to develop here the best interpretation of the experimental data so as to predict the fraction of personnel that is operationally effective after receiving full-body doses of from 1000 r to 30,000 r. The time period of interest will be the first eight hours after receipt of the dosage. In Section 2. of this Chapter, effects of lower acute doses and chronic doses will be discussed.

In the experiment of Reference 1, separate groups of four monkeys each were trained to avoid an electrical shock by taking the proper action. The animal is placed in a box which has a 100 watt lamp in the center. When this lamp lights and a buzzer sounds, the animal must run from one end of the cage to the other to avoid shock. If, however, a buzzer is sounded without lighting the lamp, the animal avoids shock by standing still. The authors of the report realized that the non-running reaction was a simpler process than the running reaction; but, it seems to us, they did not realize the full import of this difference.

Thus, the running response at the signal of light and buzzer shows not only that the animal is still thinking but that he can, or will, translate his thoughts into action. The non-running reaction, on the other hand, can indicate one of two things:

(a) That he properly interprets the signal and takes the proper action (none).

(b) That the signal does not register or that he is indifferent to it.

i.e., that for some reason he does not respond to the signal at all.

The separate groups of four monkeys each were tested on the two types of reaction prior to radiation and a base line ratio of successes to failures was recorded. The several groups were then given 1000 r, 2500 r, 5000 r, 10,000 r, 20,000 r and 30,000 r, respectively. They were then retested after irradiation. After radiation, the scores of the separate groups were uniformly higher when the non-running reaction was tested and quite sharply lower when the running reaction was tried. This indicated that two factors were involved:

1. A probability that the monkey could or would respond at all.
2. A probability that his response would be correct if he did respond.

To formulate this mathematically, let

π_2 = probability that the monkey will act in such a way as to avoid shock when the non-running signal is given.

π_1 = probability that the monkey will act so as to avoid shock when the running signal is given.

P_R = probability that the signal registers mentally and results in a decision to act, i.e., the probability that the monkey will respond.

P_S = probability that if he decides to act he will act successfully so as to avoid shock.

Now, π_1 and π_2 are given directly from the experimental data. From π_1 and π_2 we wish to calculate P_R and P_S in order to see if the peculiar behavior after irradiation is due to loss of mental function.

In order to get a successful trial in the case of π_1, the monkey must both respond to the signal and act correctly. In other words,

(1) $$\pi_1 = P_R \cdot P_S$$

In order to get a successful trial in the case of π_2, the monkey can either respond to the signal and take the correct action, or he can be indifferent or unable to respond. Mathematically this means,

$$\pi_2 = P_R \cdot P_S + (1 - P_R) \qquad (2)$$

From the actual experimental data we are given π_1 and π_2. From these we can compute P_R and P_S. The results are shown in Table 1.

The same groups of monkeys had taken the same tests prior to irradiation. In the pre-irradiation tests, the groups nearly uniformly responded to the running signal more accurately than they did to the non-running signal. Thus, the concept of response is not valid for the pre-irradiation case. Hence, we have listed P_S', the average of the running and non-running scores prior to irradiation, as an efficiency to compare with P_S.

In looking at Table 1, one should note that in two instances π_1 exceeds π_2. When this happens we have listed P_R as 1.00. The stochastic nature of the data can, of course, result in cases where P_R exceeds unity. However, one cannot fail to note two most important points about Table 1. These are:

(a) The accuracy of the monkey's decision if he can or chooses to respond is not impaired by radiation.

(b) The concept of a response probability appears valid and the failure to respond accounts for the difference between pre- and post-irradiation behavior.

To illustrate point (a) we list the average value of P_S for the four tests and compare it with the average of P_S'. The result appears in Table 2. In the cases E and F, there was only one post-irradiation test to compare with the four pre-irradiation tests, i.e, no monkeys responded after zero hours. Table 2 indicates that radiation does not impair the mental acuity of the

Table 1

Behavior of Monkey Radiation Groups After Irradiation

Dose (r)		Time of Test in Hours After Irradiation	π_1	π_2	P_a	P_s	P_s'
1000	A	0	.47	.78	.69	.68	.81
		1	.75	.80	.95	.79	.72
		4	.80	.75	1.00	.80	.74
		8	.75	.88	.87	.86	.90
2500	B	0	.30	.75	.55	.55	.79
		1	.85	.66	1.00	.83	.80
		4	.58	.83	.75	.77	.75
		8	.75	.98	.77	.97	.80
5000	C	0	.33	.90	.43	.77	.70
		1	.40	.95	.45	.89	.82
		4	.43	.88	.55	.78	.87
		8	.42	.95	.47	.89	.87
10,000	D	0	.25	1.00	.25	1.00	.62
		1	.42	.90	.52	.81	.78
		4	.48	.90	.58	.83	.78
		8	.54	1.00	.54	1.00	.82
20,000	E	0	.20	.94	.26	.77	.67
			(no response at all after zero hours)				
30,000	F	0	.19	.96	.23	.83	.81
			(no response at all after zero hours)				
20,000	E	1					.75
		4					.80
		8					.90
30,000	F	1					.76
		4					.80
		8					.92

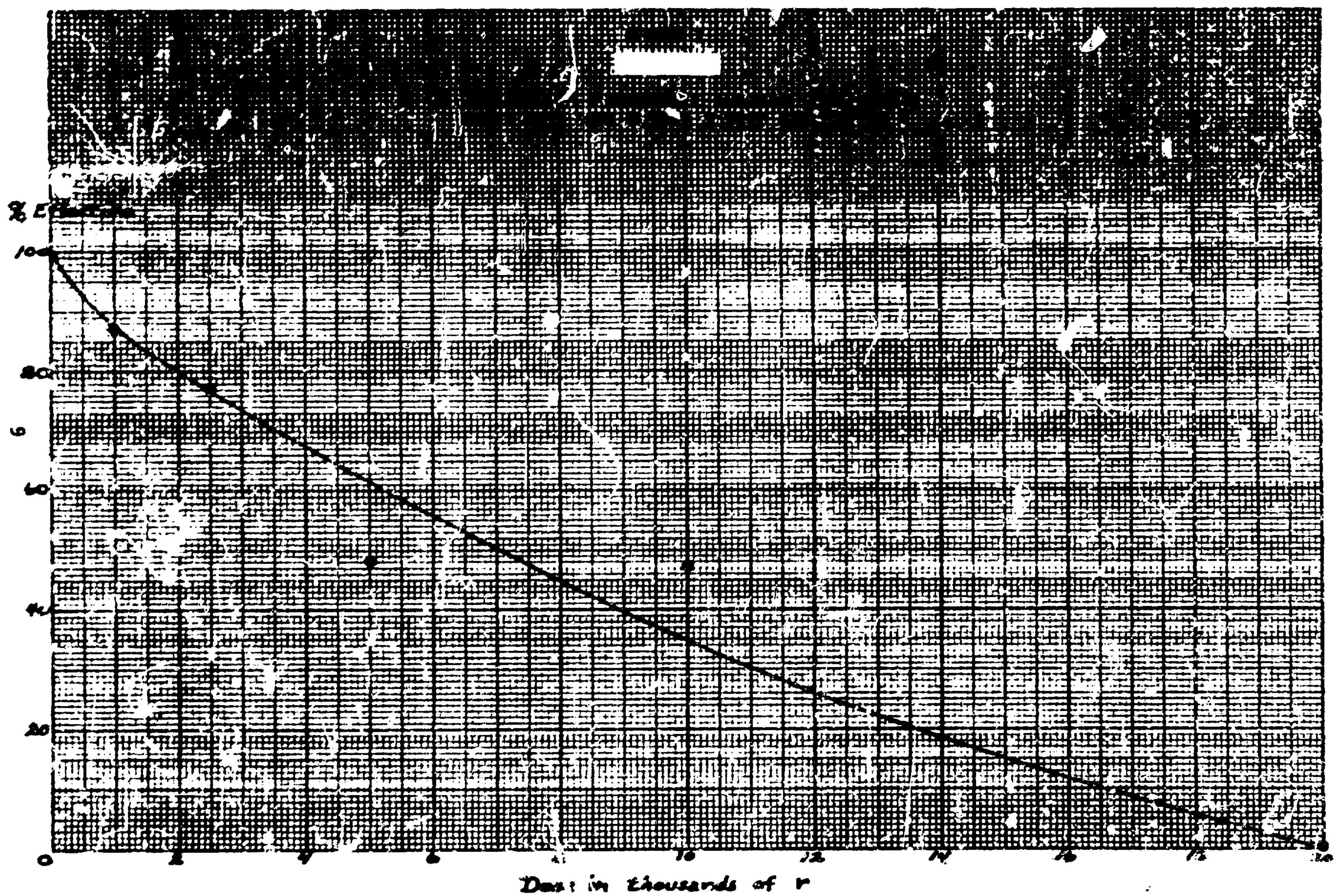
monkey. The conclusion is that if he can or will respond to stimuli, his judgment in selecting the correct course of action is unimpaired.

Table 2

The Mental Facility of the Monkey Before and After Irradiation as Measured by P_S and P_S'

Group	P_S (radiation)	P_S' (no radiation)
A	.78	.79
B	.78	.79
C	.83	.82
D	.91	.75
E	.77	.78
F	.83	.82

Although it is of philosophic interest that the monkey is unimpaired mentally, we are still faced with the fact that he does not respond. Thus, P_R of Table 1 measures the capability of the monkey after irradiation. Since P_R is relatively constant versus time over the period of 0 to 8 hours for each dose, we have plotted P_R (average) versus dose in Figure 1. In the absence of any better knowledge to the contrary, this Figure should be interpreted as giving the percentage of effective personnel versus dose in the first eight hour period. In using this Figure, it should be carefully noted that the same personnel will not necessarily be in the effective group at each time subsequent to irradiation. Thus, it was observed that although say 80% of the monkeys exposed to 2000 r were effective at time $t_1 < 8$ after irradiation, 80% would again be effective at some later time $t_2 < 8$ after irradiation but that the particular monkeys involved were not the same at the two different times. In other words, some men exposed to a dose D may be effective over one period and not at a subsequent period and vice versa.


% Effect
100
80
60
40
20
0
2
4
6
8
10
12
14
16
18
20
Dose in thousands of r

To summarize the monkey data, we can say:

1. It has been shown conclusively that the mental ability of the monkey is not impaired by high doses of radiation over the period 0 to 8 hours post irradiation. His ability or desire to translate a thought into action is the quantity which has been degraded. The ability or desire to act is measured by the probability P_R.

2. Figure 1, therefore, shows a plot of P_R versus radiation dose during the eight hour post irradiation period. The average value of P_R for the various times after irradiation is plotted in each case, e.g., for a dose of 5000 r. the averages of the numbers .43, .45, .55 and .47, as shown in Table I. is plotted as the ordinate at 5000 r.

3. One cannot say that the human and the monkey will behave the same. If we had similar experiments on say dogs, porpoises and other higher forms of mammalian life, and if these experiments all yielded the same general result, one would then have some confidence that the results would also be applicable to man. Our only conclusion is that these are the only data available and hence must be used with the hope that they apply to man.

Section 2. Acute and Chronic Effects of Radiation

Permanent Injury and Repairable Injury from Ionizing Radiation

In the discussion to follow, the term "injury" is defined in terms of the reduction in acute LD_{50} caused by a prior dose or prior doses of irradiation. Thus, for example, an animal population which has been subjected to a total dosage D (roentgens) of γ-rays can be tested at time t after cessation of the irradiation. If the LD_{50} is found to be lower by ΔD, from that of an unirradiated population sample, then ΔD is referred to as the injury at time t. Part of the residual injury ΔD disappears with time and is, therefore, referred to as the reparable part. The rest of ΔD does not disappear with time and hence represents an irreparable injury in the sense that the LD_{50} has been permanently lowered by the radiation dosage. This definition of injury has no correlation with blood count or other pathological findings. It is a measure solely of the ability of the population to withstand further irradiation.

In Reference 2., it is shown that the irreparable injury D_p and the reparable injury D_R satisfy the differential equations

2.1 $$\frac{dD_p}{dt} = f\gamma$$

2.2 $$\frac{dD_R}{dt} = (1-f)\gamma - \beta D_R$$

where $\gamma = \gamma(t)$ is the irradiation rate (in say roentgens/minute) and f is the fraction of the total dose which is irreparable. β is, of course, the exponential repair constant for the reparable injury

Now, the $\int_0^t \gamma(t)\,dt$ must be clearly understood to be the total average absorbed dose in the animal. As we shall see later, the dosage can be rather sharply attenuated throughout the body of the animal so long as the average dose is computed and then treated as though this average dose had been absorbed uniformly throughout the body of the animal.

Solving 2.1 and 2.2, one finds

$$D_p = f\int_0^t \gamma(\tau)\,d\tau$$

$$D_R = (1-f)\,\epsilon^{-\beta t}\int_0^t \gamma(\tau)\,\epsilon^{\beta\tau}\,d\tau$$

The total effective dose at any time t is now

2.3 $$D_e = D_p + D_R = f\int_0^t \gamma(\tau)\,d\tau + (1-f)\,\epsilon^{-\beta t}\int_0^t \gamma(\tau)\,\epsilon^{\beta\tau}\,d\tau$$

The expression D_e is to be interpreted precisely as follows:

Given an animal population which has been exposed to the radiation dosage $\gamma(t)$ for time t, the dose D_e, as calculated from 2.3, is exactly equivalent in mortality to the same dose D_e given instantaneously. It is assumed that the mortality for a given D_e would be determined experimentally by stopping the irradiation at time t and observing any further deaths which might occur in the next thirty days. In other words, the effective dose for chronic irradiation as calculated from 2.3 is equivalent to an acute dose of the same magnitude so far as mortality is concerned. We have no data to support this equivalence so far as how the animals "feel" is concerned.

One notes that 2.3 is easily applied to a fall-out field provided that the energy distribution of the fall-out γ's is known well enough to compute the absorbed dose or provided the absorbed dose is known empirically. Knowledge of absorbed dose would be necessary in any semi-empirical theory, however.

The justification of the empirical values of f and β is amply covered in Reference 1. and will not be repeated here. The best values from animal experiments turn out to be

$$f \simeq 0.20$$
$$\epsilon^{-\beta} \simeq 0.75$$

for all species.

The irreparable factor* is, however, the easier to measure and hence more confidence should be placed in it. $\epsilon^{-\beta}$ could conceivably be as large as 0.90; but it appears that 0.75 is a better value.

Partial Body Irradiation

Partial body irradiation experiments are of interest, not only because such exposures could occur in nuclear war, but also because they shed light on the problem of non-uniform dosage due to attenuation of the γ-ray field in the body. A theory of partial body exposure which seems to fit the observed facts is the following:

The primary damage which causes radiation death is damage to the hematopoietic system which consists of the bone marrow, lymphatic system and the spleen. If the spleen is given the proper weight, say x% of the hematopoietic system, then one may say that a dose D to a fraction y of the system is equivalent to a full-body dose of D·y. The spleen must be treated separately since it is the only highly localised portion of the system.

Appendix A gives a resume of some of the best experimental data on partial

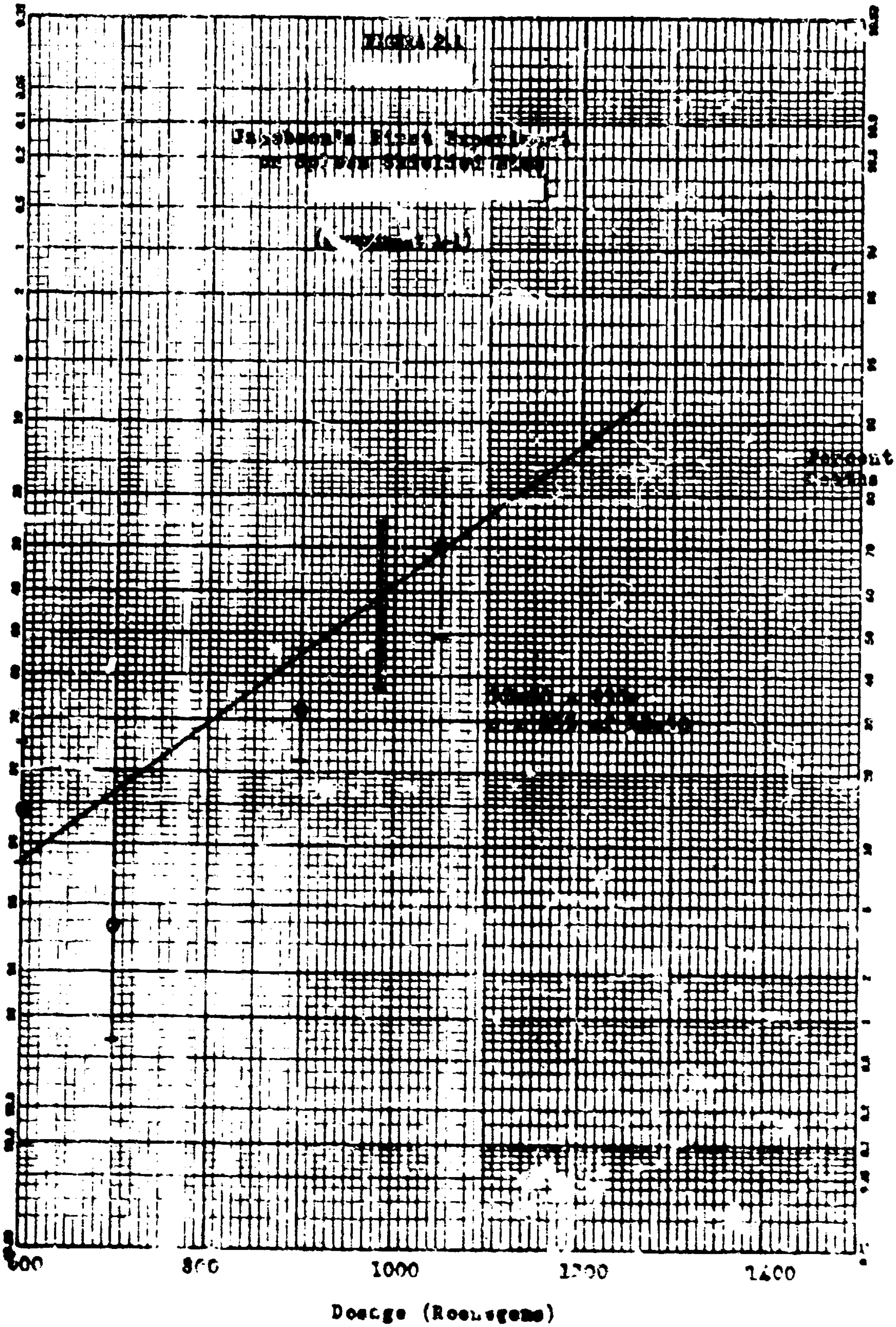
* S. Michaelson at Rochester has recently verified this value of f for dogs when the period between receipt of dose and measurement of the LD-50 was as long as nine months to one year.

body irradiation, which we have found to date. A brief discussion of the experiments of Appendix A is in order to illustrate some of the difficulties encountered in partial-body irradiation experiments and in the analysis of those experiments. There is, however, probably an important conclusion in this area.

Experiment A-1 of Appendix A gives Jacobson's original work on the effect of spleen shielding in mice. The LD-50 of the mice used in this experiment (CF-1) was stated to be 550 r. As noted in our final report of 1 March 1955, the LD-50 of mice, if taken properly, is probably in the range 700 to 800 r. Lower values are usually obtained by experimenters due to failure to measure backscatter. Hence, depending on the conditions of backscatter, a value of 550 r is acceptable and comparison with the LD-50 of spleen shielded mice is valid provided the conditions of backscatter are the same for both controls and spleen shielded mice.

Figure 2.1 gives a plot of dose versus percent deaths for spleen shielded CF-1 mice. The LD-50 now turns out to be 930 r, an increase of a factor of 1.7 over the controls. This result is, without question, statistically significant. Unfortunately, Jacobson performed a second experiment which yields significantly different results (higher effect of spleen shielding). This second experiment is hard to explain. Some comments on it will be given below.

Now, probably the best partial-body irradiation experiment which we have reviewed is that of Bond, et al, which is summarized as Experiment A-2 in Appendix A. The experiment of Bond is good because it was done very carefully. Tissue dose was measured with care and scattered dosage into the shielded regions of the animal was measured. Thus, the doses to all tissues and principal


Percent
1000
1400

organs is known.

It is interesting to attempt to explain the results of the Jacobson and Bond experiments on a simple hypothesis. Since the spleen appears to be a vital factor in radiation recovery, presumably in recovery of the haematopoietic system, let us assume that the spleen contributes a fraction f to the haematopoietic system and that the rest of the body, bone marrow and perhaps other organs, contribute a factor 1-f. If D_s is the dosage to the spleen and D_B is the dosage to the rest of the body, let us assume that the equivalent full-body dosage for the case $D_s \neq D_B$ is

2.4 $$D_e = D_s f + (1-f)\, D_B$$

If $D_s = D_B = D$, the full-body irradiation case, then $D_e = D$.

From Jacobson's experiment, when $D_s = D_B = D$, the LD-50 is 550 r. When $D_s = 0$, the LD-50 is 930 r, hence

$$930\ (1-f) = 550$$

$$\text{or } f = .41.$$

Returning now to the Bond experiment, for the case where the rat was abdominally shielded, the dosages were

$$D_s = .095\ D_A, \text{ where } D_A = \text{Air Dose}$$

$$D_B = .57\ D_A.$$

D_B here is the average dose to other body tissues. Bond gives the LD-50 of the abdominally shielded rats as 1950 r. Hence, from Eq. 2.4 using f = .41, as determined by Jacobson, one finds for D_e with D_A set equal to the observed LD-50.

$$D_e = D_A\ (.095 \times .41 + .57 \times .59) = 740 \text{ r.}$$

For Bond's second case where the abdomen is irradiated and the rest of the body shielded, one has

$$D_s = 1.03\ D_A$$

$$D_B = .52\ D_A.$$

In this case, the LD-50 turned out to be

$$\text{LD-50} = 1025\ r.$$

Thus $$D_e = D_A\ (.59 \times .52 + 1.03 \times .41).$$

Now, since $$D_A = 1025\ r$$

$$D_e = 750\ r.$$

These results are interesting since in each case the equivalent full-body LD-50 is about what Bond observes in his laboratory. He states that the LD-50 for these rats lies between 650 and 750 r.

There are, of course, many cautions which must be applied to the above analysis. First, there is some evidence that other organs, e.g., the liver, raise the LD-50 disproportionately to their fraction of body weight, when shielded. Secondly, Jacobson's later work differs from his earlier work. Comments on this will be given. Thirdly, the splenic function appears to differ in the various species. We use the word "appears" since there is considerable evidence that the apparent differences in splenic tissue in the various species dose not affect the spleen's role in radiation recovery. Much careful data study is required to resolve this question.

It is now of interest to consider Jacobson's later work (Experiment A-3). Here he finds that 78% of a sample of 135 spleen shielded mice survived 1025 r whereas less than 50% survived such a dose in the earlier experiment. The LD-50 for spleen shielded mice in this experiment was about 1100 r compared to 930 r for the previous experiment. The two results are significantly different provided that there is not a systematic dosage error (or a change in, perhaps, unmeasured backscatter) between the two experiments. Referring to Figure 2.2 which shows the data plot for spleen shielded mice in the second experiment (A-3), we see that the standard deviation is only 10% of the LD-50 value. Table 1 of A-3, however, shows that about 1% of unshielded mice exposed to 1025 r survived. This could not happen with any reasonable probability if the LD-50 (unshielded) were 550 r and the standard deviation were 10%. Hence, one suspects an error in dosage in the second Jacobson experiment.

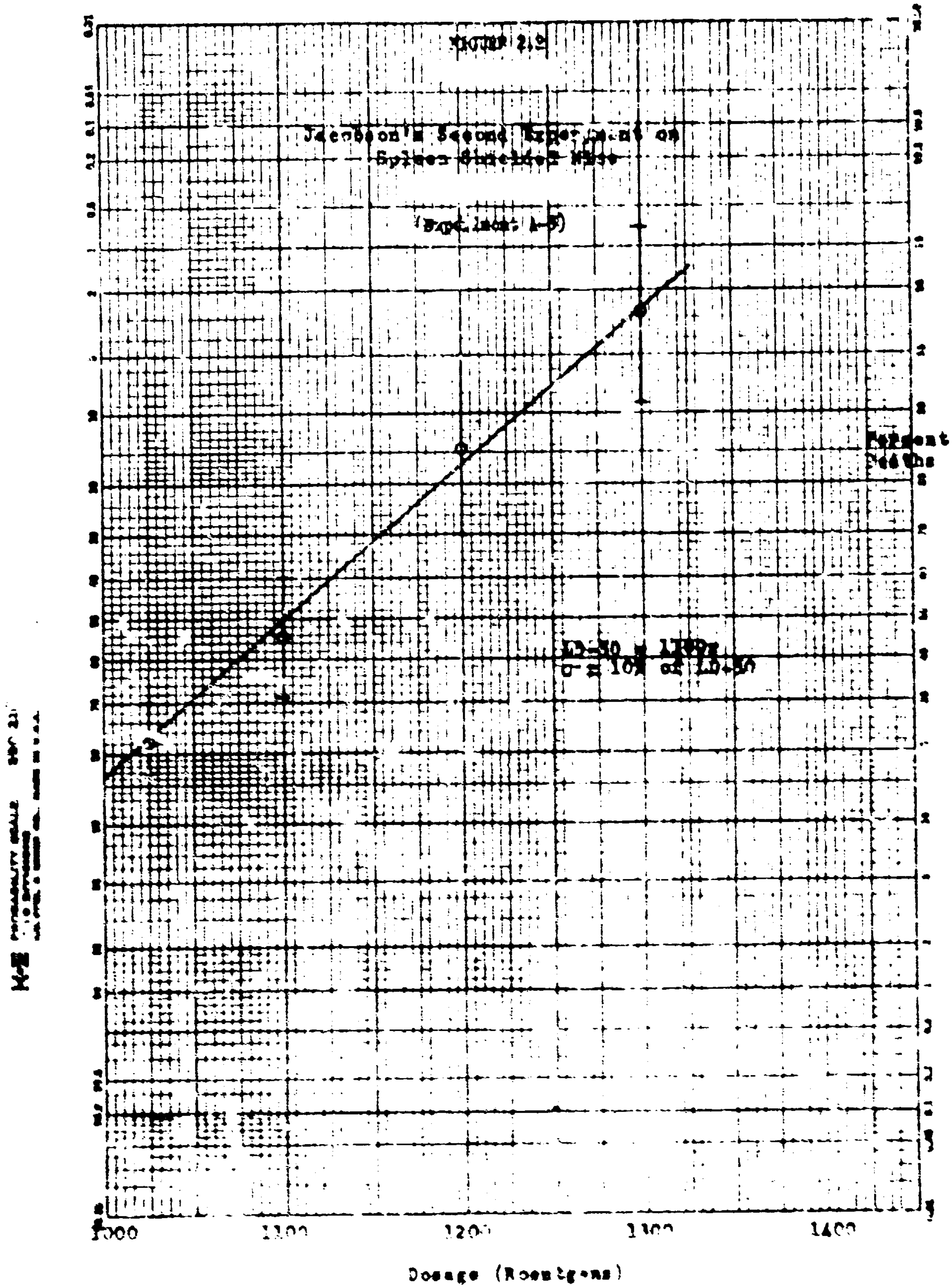
Experiment A-4 is one by Kaplan, et al, in which it appears that whereas strain A mice are effectively protected to 550 r by spleen shielding, C_{57} black mice are appreciably less protected. The data of Kaplan are, however, internally inconsistent and, it is our present belief, that little consideration should be given to this experiment.

Kaplan explains his result in terms of the presence or lack of haematopoietic activity in the spleen. However, Bacq, see Reference 3, points out that spleen shielded dogs are protected as well as Jacobson's mice and that dogs have no known splenic haematopoietic function.

We are not qualified from a biological or medical standpoint to discuss splenic tissue and function. However,* the spleen appears to take on the blood forming function in man and laboratory animals if the bone marrow fails to do so for pathological reasons. Thus, it seems that the Kaplan result is probably due to experimental error.

Thus, the dosage relationship, Eq. 2.4 is recommended for

* I am quoting the gist of a conversation with Dr. Charles Johanssen of Copenhagen, Denmark. He has been particularly interested in splenic function.


Percent Deaths
1000
1400
Dosage (Roentgens)

use in partial-body exposure. In particular, if the dose for a man in the open is attenuated from a value D_0 over the entrance surface to a value $D_0 - D$ at the exit surface, then the equivalent full-body dose should be taken as $D_0 - D/2$.

In conclusion, it should be noted that the various species rats, dogs, mice, etc. all react in the same way to irradiation so far as acute and chronic death is concerned. For example, each species can tolerate a chronic dosage of only 5 times the acute LD-50 dosage (20% retained injury for all species). Thus, one concludes that the results of this section can be reliably applied to man provided that the human acute LD-50 is known or estimated with reasonable accuracy.

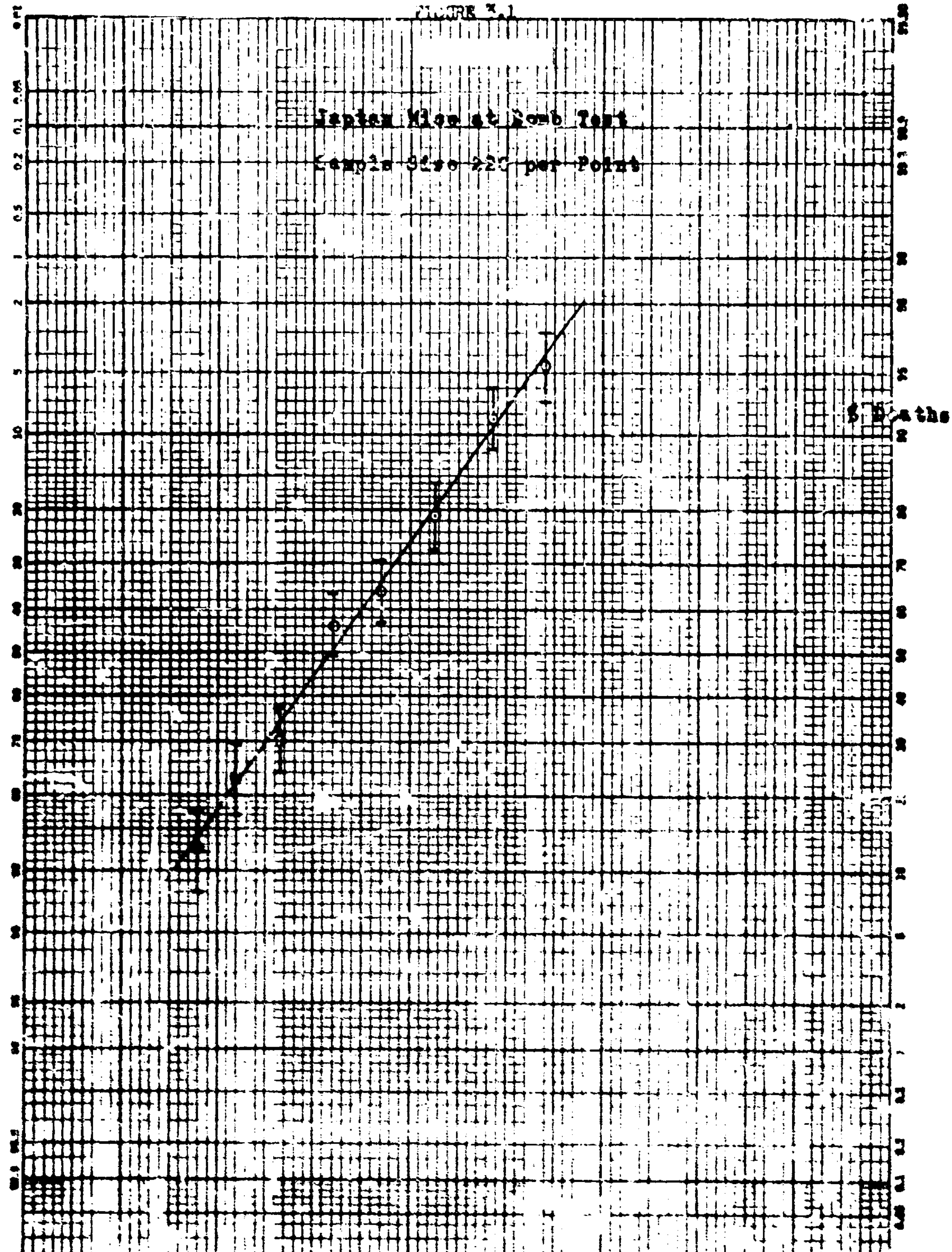
Section 3. The RBE of Radiations

In assessing the Relative Biological Effectiveness of radiations, it is necessary to be extremely careful about dosage errors. Thus, a given laboratory may make consistent measurements using 250 KVP x-rays or using Co^{60} γ-rays; but the measurements for each energy may be off by a consistent unknown error, or the errors may fluctuate due to changes of line voltage on the γ-ray tube. Sometimes the error comes from low energy backscatter which is neither recorded correctly by the instrument nor treated correctly as to attenuation in the animal. Sometimes exposures are monitored properly in phantom animals and the assumption is made that the x-ray output will not change when the real animals are introduced, whereas a sudden change in power load in the city system may alter the tube voltage and output by a sizeable factor. There are, of course, other sources of error and it is generally realized by the best experimental people that correct dosage determination is difficult, tedious and time consuming.

It is not surprising, therefore, that the determination of the RBE of Co^{60} γ-rays relative to 250 KVP x-rays sometimes leads to a factor greater than unity and, in other laboratories at other times, a factor less than unity. Moreover, from a physical standpoint, one can see no reason why those two radiations should have an RBE different from unity if the actual average absorbed dose is used in each case, provided, of course, that the attenuation of the lower energy ray is not really excessive.

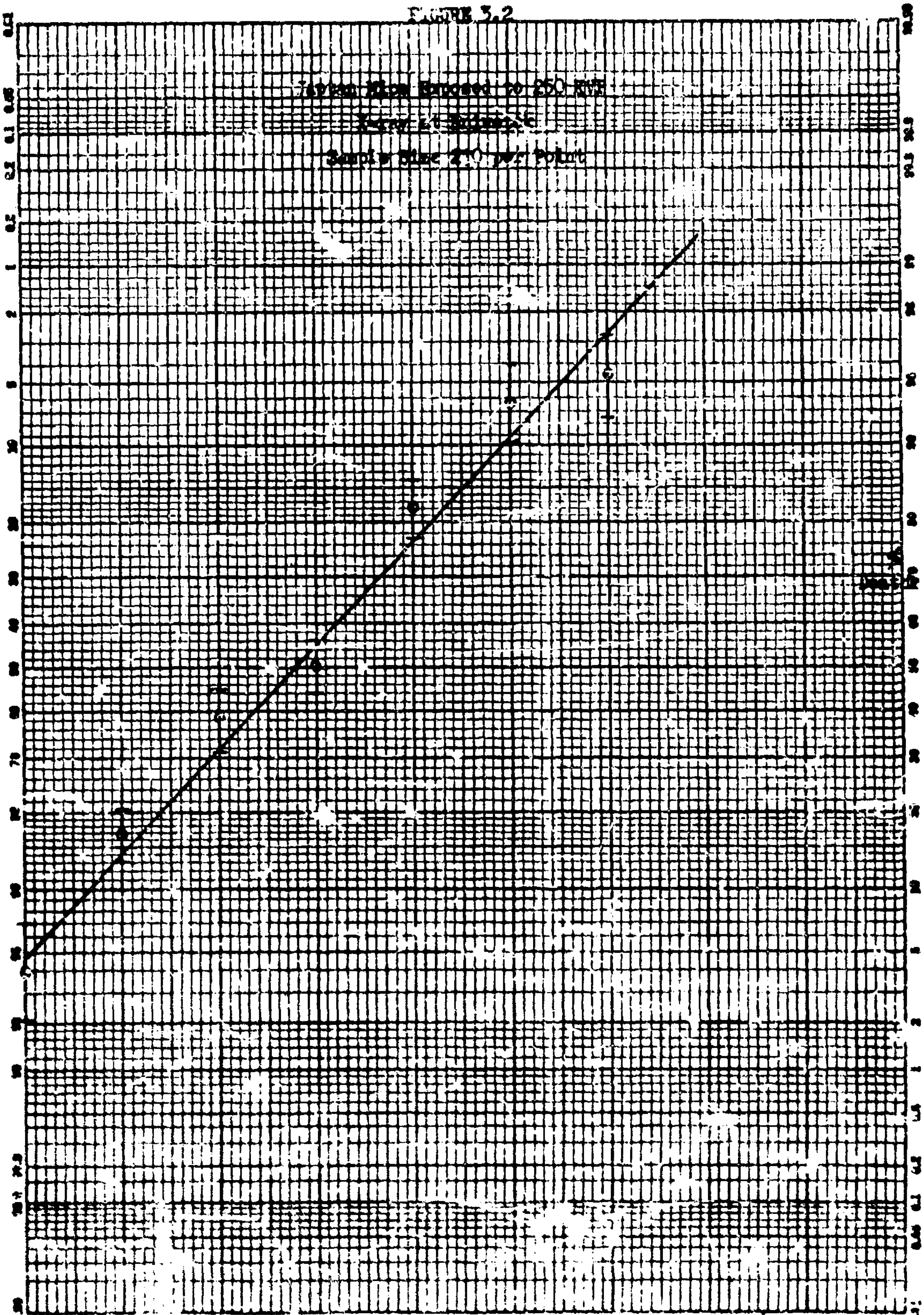
In Figures 3.1 to 3.3 we present data to show that the RBE is, in fact, unity for energies ranging from 250 KVP x-ray to 2000 KVP x-ray and to A-bomb γ's. These data were very carefully taken and the sample size per data

FIGURE 3.1



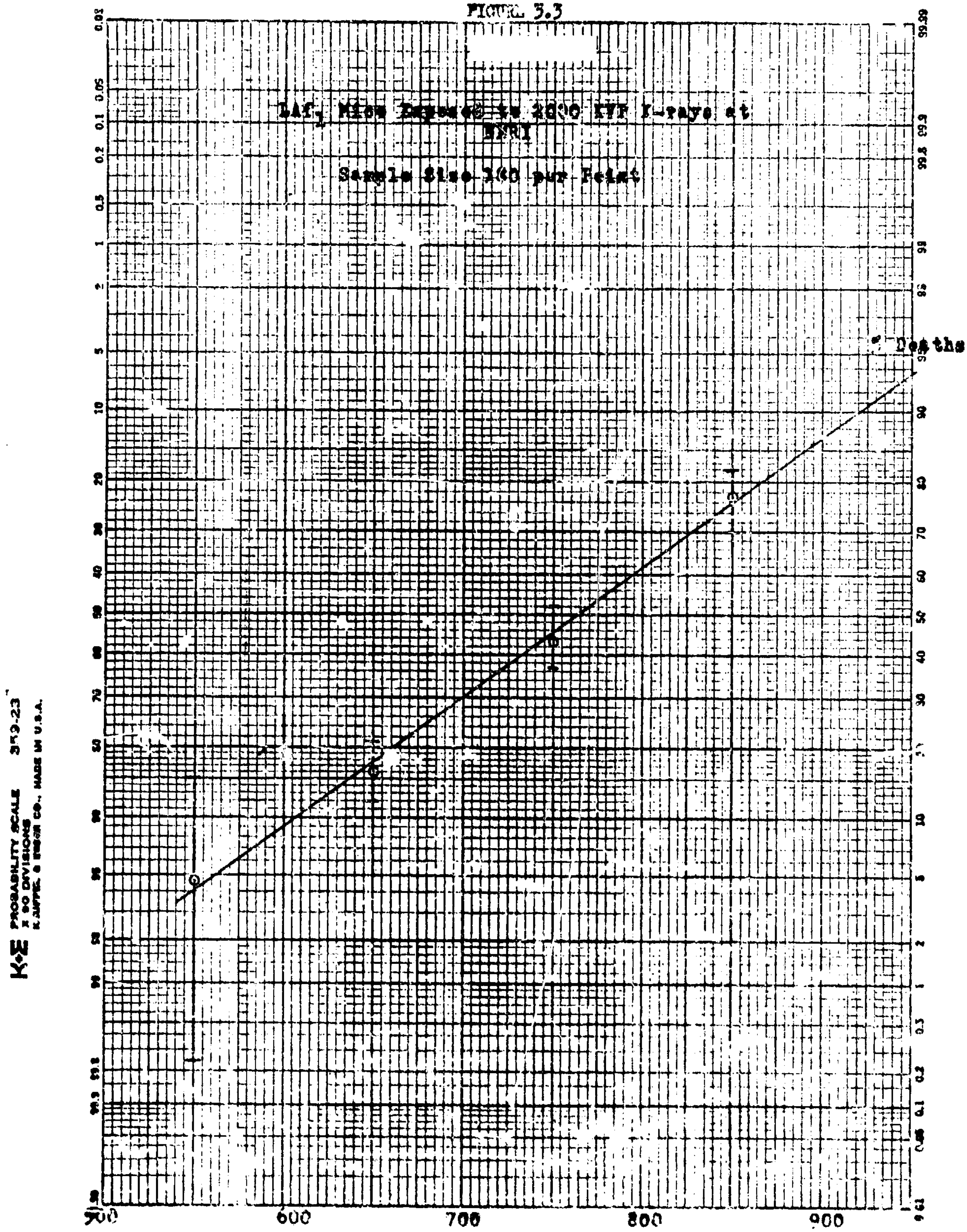
Dosage (Roentgens)

FIGURE 3.2



Dosage (Roentgens)

FIGURE 3.3



Dosage (Roentgens)

point was extremely large. One finds the following:

Table 3.1

Data on Greenhouse Mice

Strain of Mouse	Energy Source	LD-50 (roentgens)
Japtan	A-bomb γ's	760
Japtan	250 KVP x-ray	740
LAf_1	2000 KVP x-ray	765

The Japtan mice are Pacific offsprings of the LAf_1 strain and hence are essentially the same strain.

Moreover, the following careful data were taken on dogs at Greenhouse.

Table 3.2

Data on Greenhouse Dogs

Type of Dog	Energy Source	LD-50 (roentgens)
Mongrel	1000 KVP x-ray	250
Foxhounds	1000 KVP & 2000 KVP x-ray	260
Foxhounds and Mongrels	A-bomb γ's	240

Thus, again, there is no evidence of a change in RBE with energy. These Greenhouse data are presented because of the care with which they were taken. We shall refer to them later as inconsistencies appear in further data to be discussed.

The key point which we wish to discuss is the RBE of neutrons, thermal or fast, relative to x or γ-radiation. Before discussing the data, one should note that there are important physical differences between neutron effects in tissue and γ-ray effects. These differences may be briefly summarized as follows:

(a) Fast neutrons release tissue dosage mainly by ionization from recoil protons whereas the recoil electron, a much lighter particle, is responsible for γ-ray dosage. Thus, the ionization density per cm is much higher for the proton than for the electron.

(b) Neutrons which are initially thermal or become thermalized in tissue are captured by hydrogen, nitrogen and boron. The hydrogen capture leads to a 2.2 MEV γ-ray created in tissue which is quite likely to escape with no loss of energy in small animals and which is largely absorbed in very large animals. Capture in nitrogen leads to emission of a 0.62 MEV proton 95.6% of the time and to γ-radiation of average energy about 6 MEV 4.4% of the time. Thus, the proton process is the dominant one. Further, the nitrogen is changed to C^{14} and this chemical change leads to an unknown physical effect which may or may not be of importance. One knows that such a change in a nitrogen atom in DNA, for example, would be quite serious so far as that one molecule is concerned. Just how many protein molecules would have to be affected to cause a measurable physical effect is a matter for speculation. Finally, the $B^{10}(n, \alpha)Li^{7}$ reaction can occur but is of little importance due to the low boron content of the body.

From the above, one can see that, a priori, there is no reason to suspect that the neutron RBE should be unity, although the intricate nature of the processes involved does not rule out this possibility. Aside from conversion of N^{14} to C^{14}, the big difference would appear to hinge on the question as to whether the high linear energy transfer of the proton makes a significant difference.

Referring to Reference 4, we find the following summary of data on the RBE of thermal neutrons.

Table 3.3

RBE of Thermal Neutrons

Biological Response Measured	RBE (Relative to 250 KVP x-ray)
30-day survival (mice)	1.06
30-day survival (rats)	1.00
Splenic atrophy (mice)	1.06
Thymic atrophy (mice)	1.06
Intestinal atrophy (rats)	1.00
Testicular atrophy (mice)	1.88
Body Weight loss (rats)	1.00
Median Survival Time (high dose - mice)	1.50
Fe^{59} uptake (rats)	.88
Depression of mitotic activity (mice)	1.00
Leukemic "takes" (mice)	1.10 - 1.30
Shortening of mean life span (mice)	1.00

The acute effects data are, of course, of the greatest interest and, as one can see, a choice of 1.00 for the RBE is indicated. Whether the results for testicular atrophy, high dose survival time, Fe^{59} uptake, and leukemic "takes" are significant is open to question. Certainly the susceptibility of mice to leukemia is, in general, quite different from human susceptibility.

The discussion of dosage calculation and measurement in this experiment was thorough and indicates that it was done as well as one could expect.

Reference 5 leads to a fast neutron RBE of 1.7 relative to 250 KVP x-ray when the biological end point is life shortening. This is, however, not a desirable end point to use since results can be altered by disease. Further, the discussion of dosage measurement is inadequate.

To go with Reference 5, we have Reference 6 in which the RBE of bomb neutrons relative to x-rays was found to be 2.6. Although we do not wish to make undue qualifications, it should be remarked that field measurements of neutron dose have historically been questionable within at least a factor of two.

Reference 7 gives measurements of RBE of 250 KVP x-rays and fast neutrons relative to Co^{60} γ-rays. The results are as follows.

Table 3.4

RBE of x-rays and Fast Neutrons Relative to Co^{60} γ's

Energy Source	RBE
Co^{60} γ's	1.00
250 KVP x-rays	1.4
Fast Neutrons	2.0

Here we can refer to the Greenhouse data and note that the RBE of x-rays relative to Co^{60} is probably in error. In fact, results at Rochester on dogs give precisely the opposite result, i.e., an RBE of 0.7. Both results probably indicate a consistent dosage error from some such thing as unmeasured backscatter.

Reference 8 gives us another value for the RBE of fast neutrons relative to x-rays for dogs. The dosage measurements are carefully explained and appear to be quite adequate. The result turns out to be 0.8. Further, a similar measurement on dogs by another group (Ref. 9) yields a value of 0.9.

Thus, if we summarize the results of RBE of fast neutrons from References 5 through 9, we have:

Table 3.5

Summary of Measurements of RBE of Fast Neutrons Relative to 250 KVP x-rays

Reference	RBE
5	1.7
6	2.6
7	1.4
8	0.8
9	0.9

There is one further experiment which has some bearing on the matter.

Thus, in Reference 10, the RBE of α particles relative to x-rays was measured. One can, therefore, reason as follows: The RBE of fast neutrons relative to x-rays can differ from unity only if the track ionization density of the proton relative to the electron makes a difference. However, the α-particle from the $B^{10}(n,\alpha)Li^7$ reaction should have a much greater linear ionization density than the proton and hence the effect, if present for the proton, should show up even more for the α-particle. In this particular experiment, B^{10} was injected into mice and a rather uniform distribution in tissue was obtained. Radiation was done with slow neutrons. The biological end points taken were those of thymus and spleen weight loss, with the result:

	RBE relative to 250 KVP x-rays
Spleen weight loss	1.06 - 1.11
Thymus weight loss	.96 - 1.06

Thus, there is no indication that even with particles as heavy as the α-particle that the linear energy transfer affects RBE.

Summarizing, we can say the following:

(a) There is no dependence of RBE on energy for γ-rays or x-rays in the range of 250 KVP to bomb γ's.

(b) The RBE of thermal neutrons is very probably unity.

(c) The RBE of fast neutrons has been found to range from measured values as high as 2.6 to measured values as low as 0.8. The bulk of the measurements, however, are in the range 0.8 to 1.7. Thus, although we cannot say conclusively that the RBE of fast neutrons is 1.0, we can certainly say that there is not sufficient evidence to warrant a choice other than 1.0 for the RBE.

APPENDIX A

SOME PARTIAL-BODY IRRADIATION EXPERIMENTS

EXPERIMENT A-1

Reference:

The Effect of Spleen Protection on Mortality Following X-Irradiation.

Jacobson, L.O., et al.

J. Lab. Clin. Med. 34: p. 1538 1949

Abstract:

The mice used in this study were all CF-1 females and were 10 to 12 weeks of age when the experiment was initiated. The mice were kept in the animal farm in a constant temperature room (74°F) for four to six weeks before use and were maintained on a diet consisting of Derwood and water ad libitum before and after the experimental procedures described below.

Dosimetry - the X-rays administered in these experiments were generated in a 250 KV machine operating at 15 ma. A 0.25 mm Cu filter was used. The HVL in copper of the filtered beam using 240 KV was 1.0 mm. The exposures were measured with a Victoreen condenser meter equipped with a 250 r chamber. Measurements were made in air at the position occupied by the center of the animals body. The dose rate averaged 58.9 r/min at 55 cm.

Groups of mice were exposed to single doses of 600, 700, 900, 975, 1050 and 1200 r total-body x-radiation with or without lead protection of the surgically mobilized spleens.

Four groups of mice were studied. The mice in Group I were untreated controls. The mice in Group II received no irradiation but had their spleens surgically mobilized. Group III mice had their spleens surgically mobilized and during irradiation, which required from 9 to 22 minutes, depending on the dose given, their spleens were placed in paraffin boxes that offered no

appreciable shielding. The spleens of Group IV mice were placed in lead boxes with walls of 1/4 inch thickness that afforded essentially complete shielding of the spleen from irradiation. After completion of these procedures, the spleens of all groups were returned to the abdominal cavity and the incision sutured with silk. Complete recovery from the anesthetic required from 2 to 3 hours. The mice were returned to the animal farm, and deaths were recorded in 24 hour periods through 28 days.

Results are shown in Table 1.

The LD-50 for mice exposed to total-body x-radiation exclusive of the surgically mobilized lead-protected spleen is nearly twice as great as the LD-50 for mice exposed to total-body x-radiation inclusive of the spleen.

Table 1

Survival of Mice Following Single Total-Body X-Irradiation With and Without Lead Protection of the Spleen

Group	Dosage (r)	Spleen Shielded	Total No. of Mice	No. of Survivors	% Survivors
IV	600	yes	63	54	85.7
III	600	no	75	30	40
IV	700	yes	27	26	96.3
III	700	no	11	0	0
IV	900	yes	60	41	68.3
III	900	no	44	3	6.8
IV	975	yes	23	10	43.4
III	975	no	12	0	0
IV	1050	yes	23	7	30.4
III	1050	no	11	0	0
IV	1200	yes	19	0	0
II	0	operation only	64	64	100
I	0	no operation	54	52	96

EXPERIMENT A-2

Reference:

Sensitivity of Abdomen of Rat to X-Irradiation. Bond, V.P., Allen, A.C., Swift, M.N. and Fishler, M.C.

Am. J. Physiol., Vol. 161: p. 323 May 1950

Abstract:

Approximately 200 male rats bred in the USNRDL Laboratory from the Sprague-Dawley strain and weighing 250 - 300 gm. were used.

The rats to be irradiated were divided into 10 groups. Five of these groups were exposed with the abdomen shielded to 1600, 1800, 1900, 2000 and 2200 r, respectively; and five with only the abdomen exposed were given 700, 900, 1000, 1100 and 1300 r. A Picker industrial X-ray machine was used. The radiation factors were: 250 KVP; 15 ma; filter, 0.6 mm Cu; HVL, 1.3 mm Cu; Target to skin distance, 20 inches; dose rate - 47 r/min measured in air using a Victoreen 100 r thimble chamber. Results are given in Table 1. Analysis of the mortality data indicates an LD-50 of approximately 1950 r in air for rats with the abdomen shielded, and 1025 r for those receiving only abdominal exposure. The LD-50 for total-body x-irradiation is between 650 and 750 r for rats in this laboratory. The average survival times for the animals at each of several dose levels varied from 9.2 to 11.7 days and from 4.6 to 7.1 days in the 2 groups, respectively, showing no correlation with dosage in either case. No deaths occurred among control rats.

In Table 2, the rats were to be shielded either over the abdomen, or over the remainder of the body excluding the abdomen, during exposure. In order to quantitate and equate as precisely as possible the amount of radiation received

Table 1

Mortality of Rats After Partial-Body X-Irradiation

X-Ray Dose r	No of Rats Exposed	Mortality - 28 dys %	Av Time of Death days
		Abdomen Shielded	
2200	9	100	10.1
2000	15	[illegible]	9.2
1900	19	37	10.9
1800	20	30	11.7
1600	20	5	10.0
		Abdomen Only Irradiated	
1300	15	73	5.6
1100	17	59	7.1
1000	20	55	4.8
900	20	25	4.6
700	20	0	—

at any given dose level under these two conditions, it was necessary to obtain weight and organ distribution data and dosimetry measurements. To obtain the former, 4 rats were killed with chloroform and fastened in a supine position on boards in the same fashion as the rats to be irradiated. The rats were frozen with dry ice and were then sawed into transverse sections, one centimeter thick. These, identified by number, were weighed and the presence or absence of parts of major organs in each section was determined. Corresponding section weights, expressed as a percent of total body weight, and the location of organs were found to be quite uniform among the four animals studied.

Dose rates measured with a Victoreen thimble chamber in the center of the 'head', 'thorax', 'abdomen', and 'pelvic region' of a paraffin phantom shaped like a rat showed that the complex contour of the rat did not produce significant differences in tissue dose. These data are in good agreement with comparable measurements made on a dead rat. A series of dose-rate determinations was made at 3 vertical levels in a paraffin phantom throughout the length of the 'rat' using both types of shields.

It was found that if a transverse section were trisected into parts corresponding to the tissue portions into which the Victoreen meter was placed, the sum of the gram-roentgens delivered to the 3 parts was essentially the same as the product of the middle reading and the total weight of the section. Since this was so, the simpler calculation was employed. By using the latter readings and the average body weight distribution values obtained from the frozen material, it was possible to devise and place shields of such a size that the total dose in gram-roentgens received for a given dose measured in air would be approximately the same with either type of shielding. In addition, the degree of protection afforded the various organs could be estimated.

The shields were cut and moulded from lead sheets, 3.2 mm thick, either to cover or expose an abdominal area the width of the rat and 7 cm in length. The abdominal shields were calculated to cover body section 6 through 12 (xiphoid to symphysis) and the reverse shields, sections 1-5 and 13-15.

Table 2

Distribution and Quantitation of X-Ray Dosage in the Rat with Partial-Body Shielding

Section Number	Location of Organs: Intestines	Stomach	Spleen	Kidney	Adrenals	Liver	Diaphragm	Lungs	Heart	Weight of Section: Per Cent Body Weight	GM	X-Ray Dosage: Abdomen Shielded: Per Cent Air Dose	r/min	GM r/min	Abdomen Only Irradiated: Per Cent Air Dose	r/min	GM r/min
1	Head and extended portion of fore legs									12.0 (8.4 - 14.9)*	30.0	103	48	1440	6	3	90
2										5.2 (5.0 - 5.7)	13.0	102	48	625	6	3	39
3								x		5.8 (5.3 - 6.3)	14.5	101	47	682	7	3	44
4								x	x	5.3 (4.8 - 5.8)	13.3	98	46	612	10	4	53
5						x	x	x	x	5.6 (4.9 - 6.0)	14.0	87	41	574	21	10	140
6		x			x	x	x	x		7.3 (6.7 - 7.9)	18.3	20	9	165	90	43	787
7	x	x	x	x	x	x				7.4 (6.9 - 7.9)	18.5	11	5	92	101	48	888
8	x	x	x	x						7.4 (6.6 - 8.0)	18.5	8	4	74	104	49	906
9	x	x		x						7.2 (6.7 - 7.8)	18.0	7	3	54	105	50	[illegible]
10	x									6.4 (5.8 - 6.9)	16.0	8	4	64	104	49	784
11	x									5.6 (5.1 - 6.2)	14.0	11	5	70	101	48	672
12	x									[illegible] (5.0 - 6.0)	14.0	[illegible]	9	126	90	42	588
13										6.1 (5.5 - 6.4)	15.3	87	41	627	21	10	153
14										4.4 (4.0 - 4.9)	11.0	98	46	506	10	4	[illegible]
15	Tail and extended portion of hind legs									7.8 (6.5 - 9.2)	19.5	102	48	956	6	3	58
TOTAL										99.1**				[illegible]47			6146

* Range of values

** Average of 0.9 per cent of body weight lost in cutting sections.

EXPERIMENT A-3

Reference:

Further Studies on Recovery From Radiation Injury. Jacobson, L.O., et al.

J. Lab. Clin. Med., Vol. 37: p. 683 1951

Abstract:

It has been reported by Jacobson, et al, that the LD-50 of whole-body x-irradiation for intact young adult mice is around 550 r, and that only rarely will a mouse survive a dose above 800 r, whereas 40% survive a dose of 1025 r if the spleen is mobilized surgically and lead shielded during the exposure. (See Experiment A-1). These reported mortality data represent the early experience with this technique; the more recent data are shown in Table 1. These latter data indicate that with added experience with this technique fewer animals succumb and around 77.7% of mice exposed to 1025 r (with spleen shielding) survive.

The techniques for irradiating the mice for these experiments are the same as those given in Experiment A-1, except that the dose rate in this group of experiments averaged 70 r per minute at 55 centimeters as opposed to 58.9 r per minute as previously described. The animals used were all CF-1 female mice.

Additional experiments were performed in which various tissues or organs were lead shielded. The techniques were the same as those mentioned above and in Experiment A-1. The results of these experiments are given in Table 2.

Table 1

Survival of Mice Exposed to Various Dosages of X-Radiation with and Without Shielding of Surgically Exteriorized Spleen

With Lead Shielding of Spleen	Dosage in r	No. of Mice	No. of 28-day Survivors	Survival %
yes	1025	135	105	77.7
no	1025	273	3	1.1
yes	1100	42	23	54.7
no	1100	12	0	0
yes	1200	35	5	14.3
no	1200	6	0	0
yes	1300	74	2	2.7
yes	1300	26*	7	26.9
no	1300	22	0	0
yes	1600	45	0	0
no	1600	—	-	-

* Technique differed in that small artery at distal tip of spleen was not severed and thus no infarct occurred in spleen.

Table 2

Survival of Mice Exposed to 1025 r X-Radiation With Lead Shielding of Various Tissues

No. of Animals	Tissue Lead Shielded	No. of 28-day Survivors	Survival %
135	Exteriorized Spleen (0.[illegible] gm)	105	77.7
93	None	0	0
15	Exteriorized Lobe of Liver (0.8 gm)	[illegible]	33.
15	None	0	0
15	Exteriorized Intestine (2.5 gm)	4	26.[illegible]
15	None	0	0
18	Head (3.0 gm)	5	27.7
12	None	0	0
15	Right Hind Limb (incl thigh)(1.5 gm)	2	13.
15	Right Hind Foot (0.1 gm)	1	6.6
28	Exteriorized Right Kidney (0.19 gm)	0	0
8	None	0	0

EXPERIMENT A-4

Reference:

Genetic Modification of Response to Spleen Shielding in Irradiated Mice.

Kaplan, H.S. and Paull, J.

Proc. Soc. Exp. Biol. Med., Vol. 79: p. 670 1952

Abstract:

Mice of strains A (Strong) and C_{57} black, of both sexes, were divided equally with respect to age (range 32 - 69 days) among three groups. One was kept intact and received a single whole-body dose of 550 r. Physical factors were 120 KVP, 9 ma, 0.25 mm Cu plus 1.0 mm Al added filter, 30 cm mouse-target distance, 32 r/min. The spleens of the other two groups were exteriorized and placed respectively in lead or paraffin shields during irradiation to the same dose.

Results are summarized in Table 1. The earliest deaths among sham-shielded A mice occurred on the fourth day, whereas death among the intact irradiated controls were not observed until the 12th day. From this point on, however, cumulative curves of these two groups were parallel and final mortality was almost identical. A similar premature onset of death, without a greater final incidence, occurred in sham-shielded C_{57} black mice.

Table 1

Effect of Spleen-Shielding on Radiation Mortality in Two Strains of Mice

Treatment Group	32-41* days	42-51	52-61	62-71	Total		
Spleen Shielded	0/7 !	0/21	0/10	0/4	0/42	0%	Strain A
Sham	6/7	18/22	9/10	3/4	32/43	74%	
Intact	6/8	15/19	7/11	3/7	34/45	76%	
Spleen Shielded	4/7	2/11		2/17	8/35	23%	Strain C_{57} Black
Sham	6/6	11/16		5/18	22/41	54%	
Intact	8/8	14/19		3/16	26/44	59%	

* Age at time of exposure to single dose of 550 r

! Expressed as: no. dying/no. irradiated.

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II. THERMAL INJURY

Section 1. Thermal Injury to Bare Skin

The purpose of this chapter is to discuss the physical and mathematical aspects of burn injuries from nuclear weapons. First we should like to review earlier theoretical work on the problem and comment on the implications of this work.

Henriques (1) proposed that thermal injury to tissue could be explained in terms of a chemical rate process in which the protein molecules are degraded under elevated temperatures. The number of molecules degraded per unit time at a given temperature turns out to be a very critical function of the temperature itself in the range of times associated with bomb pulses. Henriques' theory fits the data reasonably well when the exposure time exceeds 30 seconds. For shorter exposure times, his theory must be replaced by a critical temperature criterion. In any event, the correlation of data which we made indicates that the concept of a critical temperature for tissue destruction is a reasonable working hypothesis in the range of bomb pulse times.

Now, as far as the physical and mathematical side of the problem is concerned, the heating of the skin by thermal flux can hardly be more complicated than a simple problem in heat conduction except for two qualifications:

1. The skin is somewhat translucent to radiation from the bomb pulse.

2. If the temperature of the skin is raised to the boiling point of water, an important change in the calculation occurs.

There have been a number of theoretical discussions of conduction of heat in the skin, e.g. (2), (3), (4). We have not, however, seen a theoretical discussion in which a reasonable mathematical approximation to the bomb pulse has been used in such a way as to get a closed mathematical result. Further, there seems to have been no mathematical discussion at all on the effect of the boiling point on skin injury. Workers at Rochester (5) have observed that if the total thermal energy Q_0 is held constant and the pulse time decreased, the depth of dermal injury goes through a maximum. They rightly conclude that the occurence of this maximum must be connected with the boiling point of the tissue water. This is so, of course, because the high value of the latent heat of vaporization of water dominates the process of heat transfer into the tissues when the boiling point is reached.

With the foregoing remarks in mind, we wish to formulate the problem of tissue (or dermal) injury taking into account these factors:

1. A realistic approximation to the bomb pulse.
2. A critical temperature for irreversible dermal injury.
3. A treatment of the problem when the boiling point of water is reached.

We shall ignore the fact that the skin is somewhat translucent to wave lengths in the bomb pulse and shall assign to the skin an effective emissivity ϵ. We shall also need values for the thermal conductivity of the skin k, the heat capacity c and the density ρ. These constants will be taken for the most part from some excellent work (2) by Dr. Thomas P. Davis.

In order to correlate the theory to be presented with experimental data we shall also need theoretical calculations using a square thermal pulse, as much of the experimental work has been done using square pulses. In this connection we shall show that a bomb pulse of peak intensity I_M is roughly equivalent to a square pulse of much lower intensity and rather long duration.

Theoretical Considerations

Let ϵ = effective emissivity of the skin

$\frac{dQ}{dt}$ = radiant intensity (cal/cm^{2}sec)

k = thermal conductivity of the skin (cal/cm$^{\circ}$C sec)

ρ = density of skin (gm/cm^3)

c = heat capacity of the skin (cal/gr$^\circ$C)

$a^2 = \rho c/k$

The first problem to be solved is the simple problem of heat conduction into the skin under a heat input of $\epsilon\, dQ/dt$. This type of problem has been discussed in so many places that it does not seem useful to repeat the derivation of the general solution here. We are considering the case, of course, where the skin is at initial temperature T_1 at time $t = 0$ (about 33°C to 35°C) and the skin surface is at $x = 0$ with $x > 0$ denoting points inside the skin. By $T(x,t)$ we shall denote the excess of the temperature over the initial temperature T_1. The solution to the problem may be written as a LaPlace transform. Thus,

(1) $$T(x,t) = \frac{1}{2\pi i}\,\frac{\epsilon}{ak}\int_{\gamma - i\infty}^{\gamma + i\infty} \frac{I(s)}{s}\,\epsilon^{-a\sqrt{s}\,x + st}\,ds$$

where $I(s)$ is the transform of dQ/dt in the t-plane, i.e.,

(2) $$I(s) = \int_0^\infty \frac{dQ}{dt}\,\epsilon^{-st}\,dt$$

The numerical treatment of the problem is easy or difficult depending on whether the direct transform (2) and the inverse transform (1) can be evaluated in closed form.

For the problem of the bomb pulse, we have noted that functions of the form

$$dQ/dt = \text{constant} \cdot t^{-n/2}\,\epsilon^{-K^2/4t}$$

are convenient analytically and that, in particular, the function

(3) $$\frac{dQ}{dt} = \frac{Q_0}{4\sqrt{\pi}}\,K^3 t^{-5/2}\,\epsilon^{-K^2/4t}$$

gives an adequate approximation to the bomb pulse. We associate K with the time at which dQ/dt reaches a maximum and find

(4) $$K^2 = 10\,t_m$$

where t_m is the time at which dQ/dt reaches the maximum.

If (3) is used for dQ/dt in (2) and (1), the temperature distribution turns out to be

$$T(x,t) = \frac{\epsilon Q}{ka\sqrt{\pi t}} \left[\frac{K(K+ax)}{2t} + 1 \right] \epsilon^{-\frac{(K+ax)^2}{4t}} \tag{5}$$

The maximum temperature at any depth x occurs at time

$$t = t_s = \frac{K+ax}{2} \left[\sqrt{2K^2 + \frac{a^2x^2}{4}} - \left(K - \frac{ax}{2}\right) \right] \tag{6}$$

In particular, the maximum temperature at x = 0, the skin surface occurs when

$$t_s = 5t_m(\sqrt{2} - 1) \tag{7}$$

We are interested in this last value of time since boiling occurs at x = o first.

Now, for the square wave pulse where

$$\frac{dQ}{dt} = \frac{Q_0}{t_0} \qquad 0 \le t \le t_0$$

and $dQ/dt = 0$, $t > t_0$, the solution for $T(x,t)$ turns out to be

$$T(x,t) = \frac{\epsilon Q_0}{kat_0} \left\{ 2\sqrt{\frac{t}{\pi}}\, \epsilon^{-\frac{a^2x^2}{4t}} - ax\, \mathcal{E}fc\left(\frac{ax}{2\sqrt{t}}\right) \right\} \tag{8}$$

where $\quad \mathcal{E}fc(u) = 1 - \frac{2}{\sqrt{\pi}} \int_0^u \epsilon^{-t^2}\, dt \qquad$ and $\qquad 0 \le t \le t_0$

For the square wave, the solution changes after $t = t_0$ since the radiant intensity stops abruptly at that point. We can note at this point that the solution for $t > t_0$ is

$$\begin{aligned} T(x,t) = {} & \frac{\epsilon Q_0}{kat_0} \left\{ 2\sqrt{\frac{t}{\pi}}\, e^{-\frac{a^2x^2}{4t}} - ax\, \mathcal{E}fc\left(\frac{ax}{2\sqrt{t}}\right) \right\} \\ & - \frac{\epsilon Q_0}{kat_0} \left\{ 2\sqrt{\frac{t-t_0}{\pi}}\, e^{-\frac{a^2x^2}{4(t-t_0)}} - ax\, \mathcal{E}fc\left(\frac{ax}{2\sqrt{t-t_0}}\right) \right\} \end{aligned} \tag{9}$$

<u>Values of the Skin Constants</u>

The thermal conductivity of water at the temperatures in question is about 1.65×10^{-4} cal/cm sec °C. Since the skin is [illegible] 5 % water and since the thermal conductivity of the fibrous tissues must be rather low, one could estimate the conductivity of the skin at $0.65 \times 1.65 \times 10^{-4}$ which is approximately 10^{-3} cal/cm sec $^{\circ}$C. This is the value which Davis has measured. Thus, one can be fairly confident of this number. Further, Davis measures ρc to be 1.12 cal/cm^3 $^{\circ}$C and this value is reasonable. For the effective emissivity, a value of $\epsilon \approx 0.4$ is reasonable. One would expect an ϵ of about 0.9 if the skin were opaque. The lower value apparently is caused by back scatter of radiation from small depths near the surface.

Before proceeding with the alteration of the theory when the boiling point is reached, we wish to indicate how the theory derived so far will be applied to problems of burns. It will be shown that there is a critical temperature T_c which results in irreversible dermal damage and that the value of T_c can be derived from the experimental data. For the bomb pulse, the depth of burn will be the deepest point at which $T(x,t_s)$ as given by (5) exceeds T_c, under the condition that the surface temperature never exceeds the boiling point. A similar process can be followed for the square pulse data. We will correlate the theory both with bomb pulse data and with square pulse data and show that the proper square pulse simulates a bomb pulse adequately.

The best data on the time at which the boiling point is reached are data using square pulses. Hence we shall have to use square pulse data in this case for correlation with the theory.

Let us note for reference that the maximum surface temperature for a square pulse occurs at $t = t_o$ and this temperature is from (8), setting $x = 0$

(10) $$T(0,t_o) = \frac{2\epsilon Q_o}{k a \sqrt{\pi t_o}}$$

The maximum surface temperature for the bomb pulse is obtained from (5) by substituting t_s from (7) and setting $x = 0$. This turns out to be

(11) $$T(0,t_s) = \frac{1.03\,\epsilon Q_o}{k a \sqrt{\pi t_s}}$$

or $$T(0,t_m) = \frac{.716\,\epsilon Q_0}{ka\sqrt{\pi t_m}}$$ since $$t_s = 2.07\, t_m$$

One notes that (10) and (11) will agree only if the duration of the square pulse is chosen properly in regard to $t_s \approx 5\, t_m (\sqrt{2} - 1)$. Thus, one must choose

$$\frac{2}{\sqrt{t_0}} = \frac{.71}{\sqrt{t_m}} \qquad \text{or} \qquad t_0 = 7.85\, t_m$$

Thus, to simulate the bomb pulse, the square pulse must have a duration of about 8 times the time to the maximum of the bomb pulse.

The peak intensity (constant) in the square pulse must be correspondingly reduced below the peak intensity in the bomb pulse.

Again one notes from either (10) or (11) that if the peak temperature is set at the boiling point of water, each equation predicts that ϵQ_0 will vary as the square root of the time parameter. We shall see later that the experimental data confirm this prediction.

The Boiling Point Problem

1. The Square Wave.

Returning to Eq. (8), we note that boiling starts at the surface when $T(0,t) = T_0$, where T_0 is the difference between the initial skin temperature and the boiling point temperature. Thus, under ordinary thermal conditions, $T_0 \approx 65°C$ and the boiling point is reached when

(11a) $$T_0 = \frac{2\epsilon Q_0}{ka t_0}\sqrt{\frac{t}{\pi}}$$

It is evident from a physical standpoint that when the boiling point is reached at the surface of the skin it becomes quite difficult to increase the depth of burn. The high value of the latent heat of vaporization (538 cal/gr) creates a heat sink which is large compared to the energy required to heat to the boiling point. Thus, at approximately the time that the boiling point is reached one would expect the maximum depth of dermal injury to occur*. To be sure, the

* For fixed total energy Q_0, of course, under variation of the pulse time t_0.

maximum damage can occur at appreciably later times if the remaining heat flux is large enough.

To discuss the effect of the boiling away of skin water, one must start with some reasonable assumptions. These would appear to be:

1. The skin water boils in a thin layer near the surface and recondenses at the termination of the heat pulse.

2. When the skin surface reaches the boiling point, the temperature distribution in the skin does not differ significantly from what it would have been if the surface temperature had been held at the boiling point for the same length of time. This assumption is justified by actual calculations to be given later and the error involved in the assumption is indicated.

3. The depth of the boiling zone is small compared to the total depth of the injury. Thus, the problem can be treated as one in which the skin surface is held at $T = T_0$ (T_0 - temperature rise required to reach the boiling point) after boiling is reached resulting in the usual conductive solution for such problems. Thus

$$T_1(x,t) = T_0 \,\mathrm{erfc}\left(\frac{ax}{2\sqrt{t}}\right) \tag{12}$$

4. There is a critical temperature $T = T_c$ at which tissue necrosis occurs.

It is clear, of course, that assumption (1) cannot hold under extreme conditions of heat input. Thus, for large ϵQ_0, as t_0 is decreased, one ultimately reaches a point at which steam is lost from the surface. Without knowing a great deal about mechanical and thermal properties of the skin, we cannot treat the steam loss problem except by appealing to the data given in Reference 5; e.g., see Figure 8 of this Chapter.

We do know, however, that when the boiling point is reached, the temperature distribution shifts from that given by (8) to that given by assumption (4). This gives us a maximum depth of damage which corresponds to setting $T = T_c$ in (12) with $t = t_c$. t_o is, of course, determined from (11a), for square pulses. After boiling is reached, the depth of damage then varies as given by (12), i.e., as

$$\text{(13)} \qquad \operatorname{Erfc}\left(\frac{ax}{2\sqrt{t_c}}\right) = \frac{T_c}{T_o}$$

It is clear that under the assumptions made, (13) represents a maximum in the expected depth of damage since it takes some time to shift from the original temperature distribution to that given by (12). t_e is the time at which recondensation is complete. This time will be calculated using reasonable assumptions. One notes that x decreases as $\sqrt{t_c}$.

In the case of very small t_o, the skin water must boil away completely to some depth δ. This case is not a very important one as it is an extreme case.

To calculate the general expression to t_e, we proceed as follows. The calculation method is illustrated for a square pulse. Now, for the square pulse one has

$$\text{(14)} \qquad T(0,t) = \frac{2\epsilon Q_o}{k a t \sqrt{\pi}} \sqrt{t}$$

Boiling is reached at $t = t_B$ given by inserting $T(0,t) = T_o$ in (14). After boiling is reached, the temperature distribution is given by

$$T = T_o \operatorname{Erfc}\left(\frac{ax}{2\sqrt{t}}\right)$$

which yields

$$-k \left.\frac{\partial T}{\partial x}\right|_{x=0} = \frac{k T_o a}{\sqrt{\pi t}}$$

For a square pulse, the heat in the boiled fluid is $\epsilon Q_o(1-t_B/t_o)$ and at the time t_e at which recondensation is complete one must have

$$\text{(15)} \qquad \int_{t_B}^{t_e} \frac{k T_o a}{\sqrt{\pi t}}\, dt = -\int_{t_B}^{t_e} k \left.\frac{\partial T}{\partial x}\right|_{x=0} dt = \epsilon Q_o \left(1 - \frac{t_B}{t_o}\right)$$

The depth of damage at $t = t_e$ is then given by

$$x = \frac{2\sqrt{t_e}}{a} \, \mathrm{Erfc}^{-1}\left(\frac{T}{T_0}\right) \tag{16}$$

Integrating (15) one has

$$\frac{2kT_0 a}{\sqrt{\pi}}\left(\sqrt{t_e} - \sqrt{t_B}\right) = \epsilon Q_0 \left(1 - \frac{t_B}{t_e}\right) \tag{17}$$

with

$$\sqrt{t_B} = \frac{k a t_k \sqrt{\pi}}{2 \epsilon Q_0} T_0 \tag{18}$$

from (14). Thus, equations (16), (17) and (18) give us the required solution.

As mentioned before, this solution holds so long as the vaporized water re-condenses in steam blebs. At extreme conditions, the steam explodes from the surface. When this occurs, the depth of damage falls drastically and can rise again only at heat inputs so high that they are of little interest here.

To summarize the calculations:

1. Square Pulses.

The maximum depth of damage occurs approximately at the boiling point. At this point ϵQ_0 varies with $\sqrt{t_0}$ as given by

$$\epsilon Q_0 = \frac{k a T_0 \sqrt{\pi t_k}}{2}$$

After the boiling point is reached and in the case of no steam loss, the depth of damage is given by (16) along with (17) and (18).

2. Bomb Pulses.

The maximum depth of damage again occurs approximately at the boiling point. At this point ϵQ_0 varies with $\sqrt{t_m}$ as given by (11) with $T(0, t_B)$ set equal to T_0.

After the boiling point is reached, the depth of damage is given by (16) along with (17) except that the right hand member of (17) must be changed to read

$$\epsilon Q_0 - \int_0^{t_B} \frac{dQ}{dt} dt = \epsilon Q_0 \, [illegible] \left(2 \cos \frac{t}{t_B} \ldots \right)$$

where I is the incomplete Gamma function as tabulated by Pearson. For the bomb pulse, it is clear that (18) must be replaced by (5) with $T(0,t) = T_0$ and $t = t_B$ to be determined.

The Critical Temperature

Henriques (1) has discussed the problem of tissue destruction in terms of a simple first-order reaction rate formula. For constant tissue temperature, Henriques hypothesizes that tissue destruction follows the law

$$\Omega_c = Pt\,\epsilon^{-\frac{\Delta E}{RT}} \tag{19}$$

ΔE is the activation energy of some unspecified tissue destroying reaction, P is a constant, t = time required for tissue destruction, T = temperature and R = gas constant. Ω_c is some fixed critical value required for tissue necrosis. If one plots Log t versus 1/T, and if the theory is correct, one should obtain a straight line. Henriques does obtain a straight line for $t \geq 30$ seconds but the data fit is poor below this point. We wish to demonstrate that, although Henriques' theory may be correct for large T, the tissue destruction criterion (19) is not correct for times in the bomb pulse range. In this range tissue destruction occurs at some critical temperature $T = T_c$ independent of pulse time. To show that this is so, we appeal to the basic data.

Now, Henriques' data were taken by holding the skin surface temperature at some fixed value T_0 for time t_0. For each T_0 there is, of course, a time at which the injury corresponds to transepidermal necrosis with damage limited to the epidermis only (a thickness of about 0.1 mm). The various values of T_0 and t_0, corresponding to the above described injury, were determined by Henriques.

Looking at the problem mathematically, the temperature distribution is

$$T = T_0\,\mathrm{Erfc}\left(\frac{x}{2\sqrt{\kappa t_0}}\right) \tag{20}$$

Now, for $t_0 \geq 1$ and $x \approx .01$ cm, $ax/2\sqrt{t_0}$ is small so that

$$Erfc\left(\frac{ax}{2\sqrt{t_0}}\right) \simeq 1 - \frac{ax}{\sqrt{\pi t_0}}$$

and

$$Log\left[Erfc\left(\frac{ax}{2\sqrt{t_0}}\right)\right] \simeq -\frac{ax}{\sqrt{\pi t_0}}$$

Thus, from (20),

(21) $$Log\,T = Log\,T_c - \frac{ax}{\sqrt{\pi t_0}}$$

where x is the epidermal thickness and T is the temperature of the epidermis. Clearly, there is a critical temperature $T = T_c$ if when one plots Log T_0 versus $t_0^{-\frac{1}{2}}$ a straight line is obtained. Figure 1. shows the result of plotting Henriques' data in this way. The body skin temperature of the pig has been taken to be 34°C and the temperature plotted is, of course, the elevation in temperature above the normal temperature of 34°C. One notes that the plot is quite linear up to times of 25 seconds. The intercept at $t_0^{-\frac{1}{2}} = 0$ yields the critical temperature $T_c = 18°C$ above the normal temperature of 34°C. Thus, the absolute critical temperature is 52°C. Further, the slope of the curve is 0.24 and this should correspond to $ax/\sqrt{\pi}$. Setting a = 34.4, this yields x = .012 cm which is certainly a reasonable value for the epidermal thickness. Thus, Henriques' data clearly indicates a critical temperature for tissue destruction in the range of bomb pulse times.

Looking at further data, Kuhl, Sheline and Alpen give the following data for heating of the rat ear with hot water. The injury criterion is blister formation which corresponds roughly, in this case, with transepidermal necrosis. The data are given in the table below. Unfortunately, we do not know the initial temperature of the epidermis. In an area such as the ear, however, the epidermal temperature could hardly be more than about two degrees above the room temperature of 25°C. The temperature at the center of the ear (... below surface) was

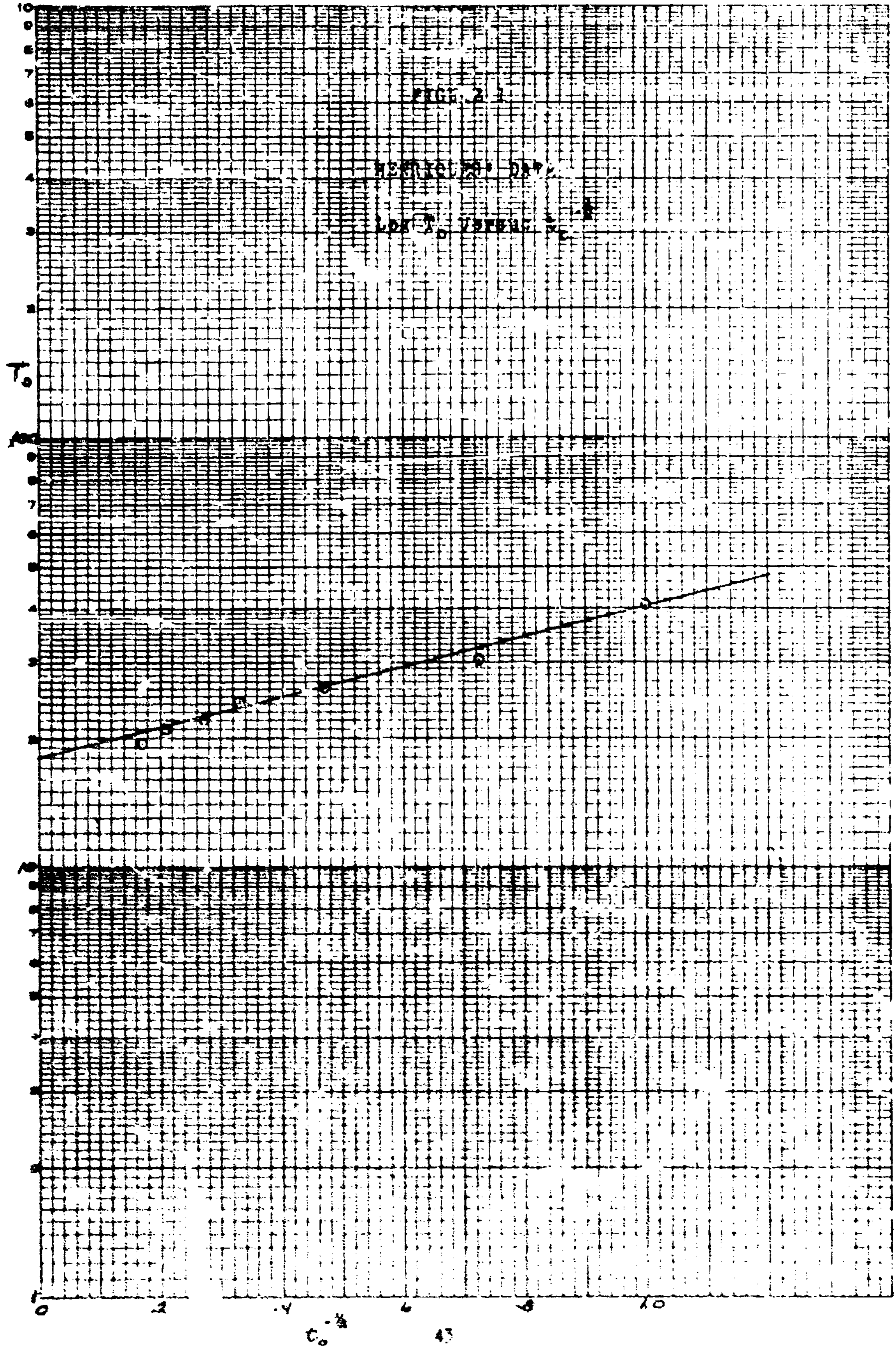
FIG. 2.1
HENDRICKS' DATA
Log T_o Versus $C_o^{-1/3}$
T_o
0
.2
.4
.6
.8
1.0
$C_o^{-1/3}$

about 29°C. Thus, an epidermal temperature of 25°C is probably about correct. The plot of the Alpen data is shown in Figure 2. Note that the point at 300 seconds cannot be used as this length exposure puts us in the range where the Henriques' theory is valid. The point at 0.25 seconds is suspect due to the difficulty of controlling the exposure time.

Table 1

Median Effective Water Temperatures
Required to Produce Blistering

Exposure Time (sec)	ET_{50} Exposed Side of Ear °C
0.25	71.0 ± 1.1
0.50	69.0 ± 0.2
1.0	64.4 ± 0.2
20.0	53.0 ± 0.2
300.0	47.6 ± 0.?

The critical temperature turns out to be 51°C as against 52°C from the Henriques data. Thus, it appears that we can talk about a critical temperature and that the value of this critical temperature is about 52°C.

The reader should note that the slope of the curve is 0.23 corresponding to $x = 0.12$ mm. This confirms that we are again talking about transepidermal necrosis. The authors surmised that this was so.

Comparison of Theory with Measurements

We have noted from Eqs. (10) and (11) that a bomb pulse may be replaced by an equivalent square pulse in the sense that t_o for the square pulse may be adjusted so that if $t_o = 7.85\ t_m$, the maximum bomb pulse surface temperature and the maximum square pulse surface temperature are the same. From a practical standpoint, however, this equivalence may be extended as follows:

(a) If the time to the maximum of the bomb pulse is t_m and if the bomb

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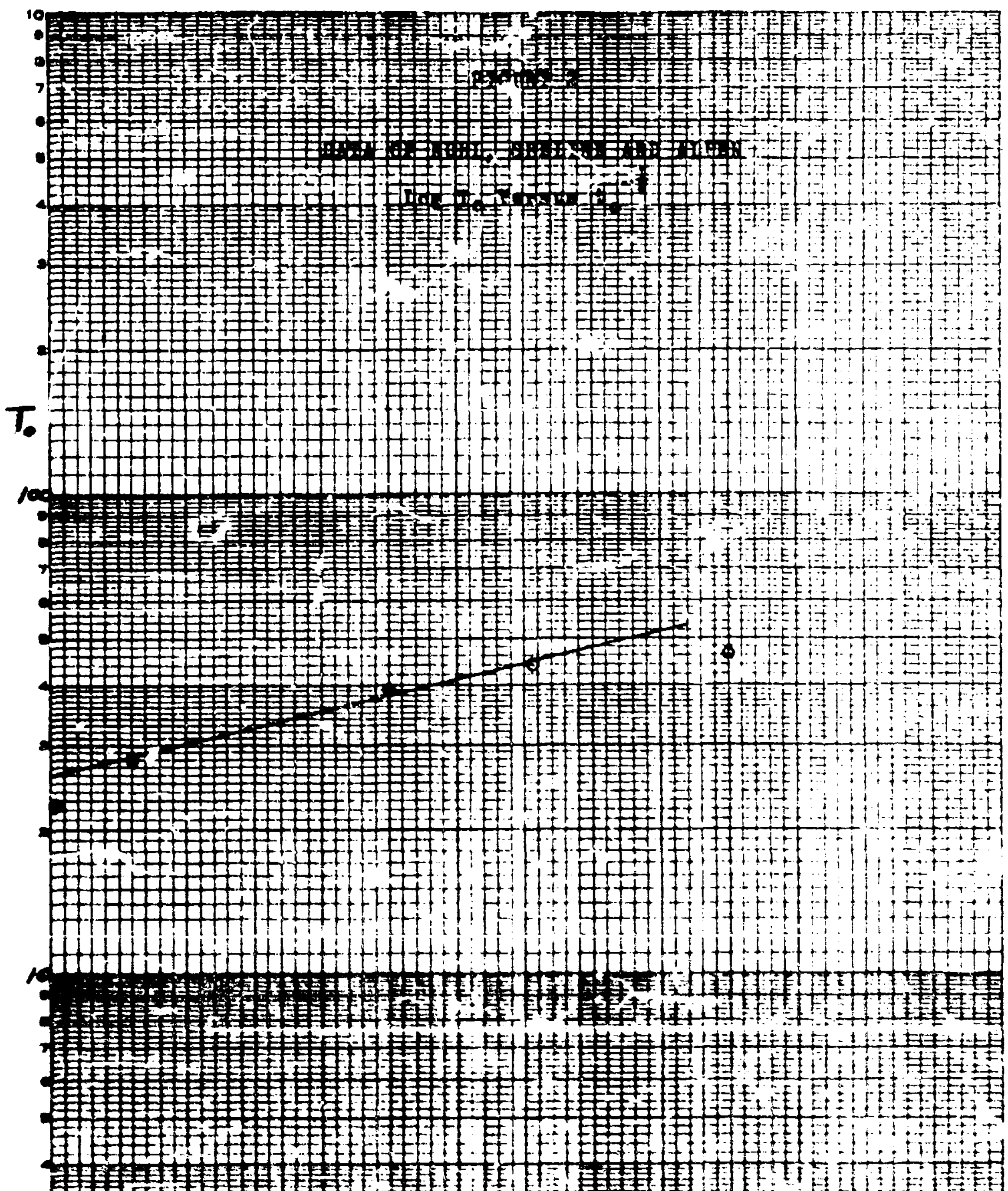


pulse is replaced by a square pulse of duration 7.85 t_m, then the maximum depth of burn will be given to good approximation by

$$(22) \qquad T(x, 7.85\, t_m) = T_c$$

where $T(x, 7.85\ t_m)$ is the square pulse temperature distribution at $t = 7.85\ t_m$ (the end of the pulse), T_c is the critical temperature and x is the depth of burn as calculated from (22). In other words, the temperature distribution at the end of the square pulse does not yield a significantly different depth of damage than one would obtain by finding the maximum depth at which T reaches T_c for the bomb pulse itself. This approximation is interesting and useful and will be justified by actual calculation.

A second point to note is that the square pulse-bomb pulse equivalence as stated above carries over into the region where the surface temperature reaches the boiling point.

Thus, in comparing theoretical results with experimental results we shall compare square pulse calculations with square pulse experimental data. Then we shall compare bomb pulse data with the square pulse equivalent calculation as stated above. Finally we shall show that the bomb pulse theoretical calculations do not differ significantly from the equivalent square pulse theory as defined by (a) above.

In the previous section, we determined the critical temperature T_c, and the skin constants ϵ, ρ, c and K were determined from Reference (2). We now use Reference (5) to compare square pulse data with square pulse theory and Reference (6) to compare equivalent square pulse theory with both bomb pulse data and bomb pulse theory.

For the convenience of the reader we will present some plots of the basic theoretical expressions. Figure 3 shows a plot of

$$\frac{T(x,t_o)\sqrt{t_o}}{\epsilon Q_o}$$

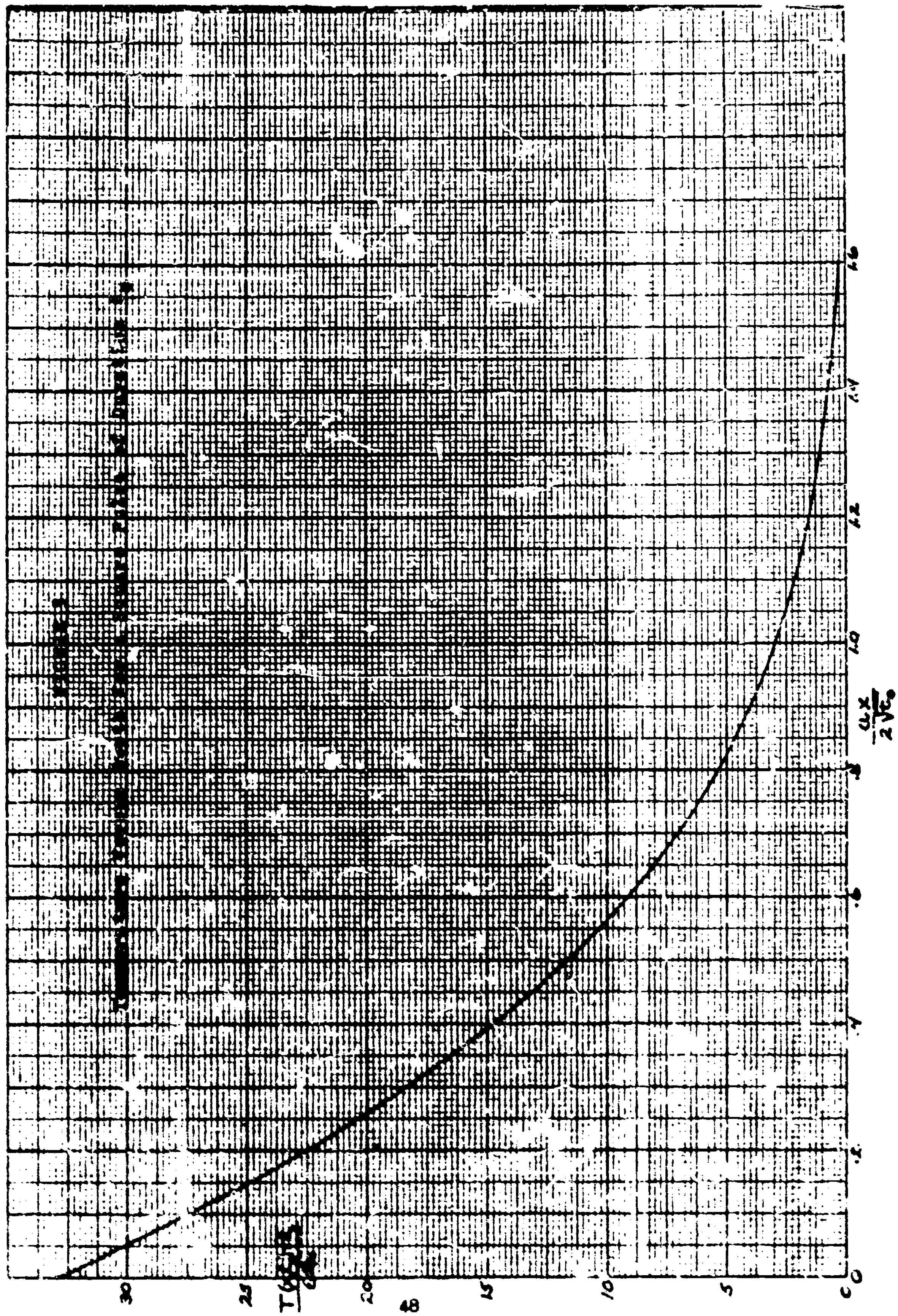
for the square pulse. $T(x,t_o)$ is, of course, the maximum temperature achieved at any depth x. If one sets $T(x,t_o) = T_c$, then one can calculate the depth of damage from this curve provided, of course, that $T(0,t_o) \leq T_o$, i.e., provided boiling is not reached at the surface.

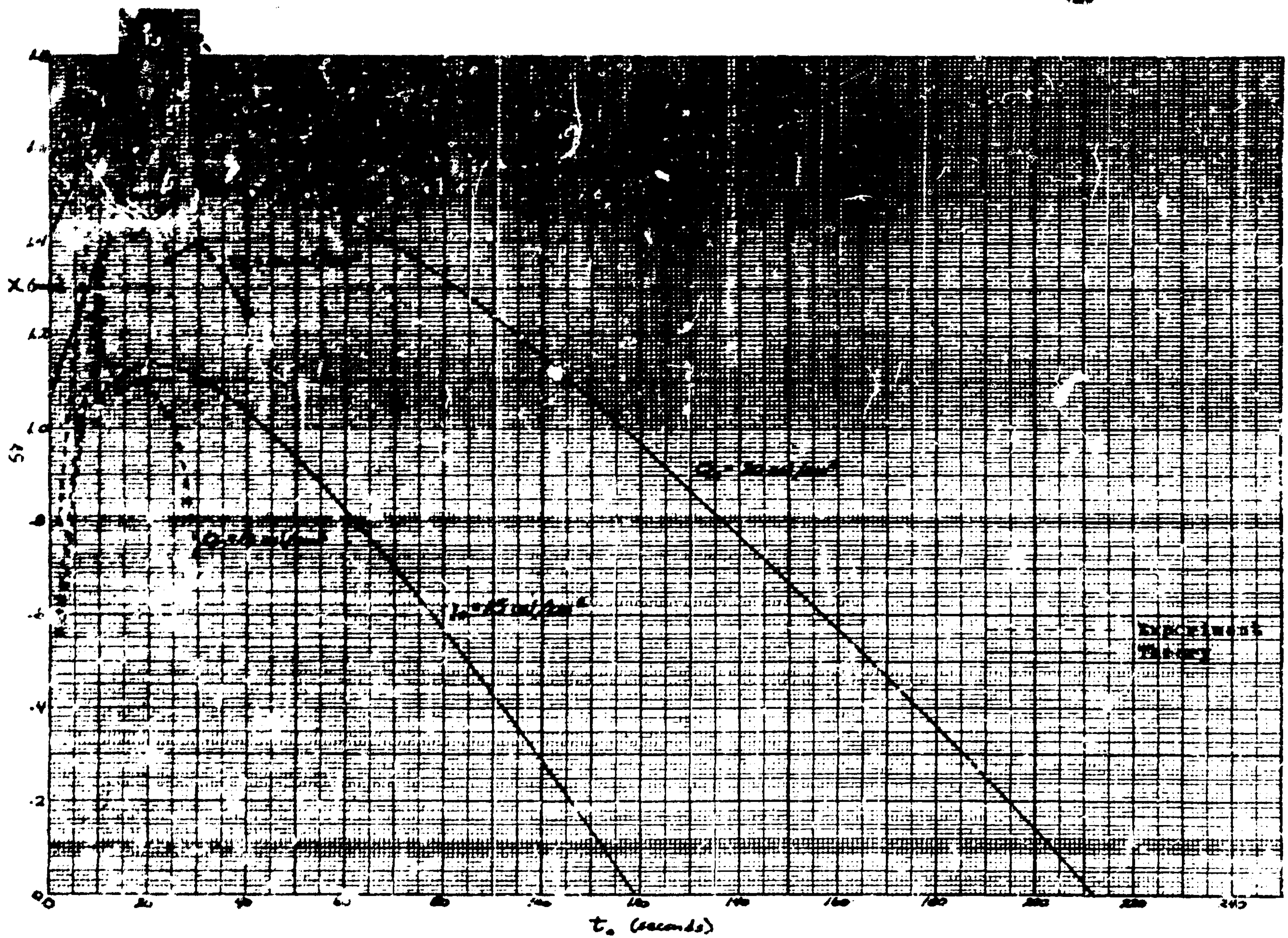
Curves of depth of damage versus t_o for fixed Q_o are shown in Figures 4 and 5 for a critical temperature of 52°C. The damage depth after boiling sets in is calculated as indicated earlier. The sudden jump at the boiling point is caused, of course, by the assumption that the temperature distribution shifts immediately to that given by (12). This does not occur, of course, except at shallow depths and hence the calculations for the boiling case represent the greatest depth of damage to be expected.

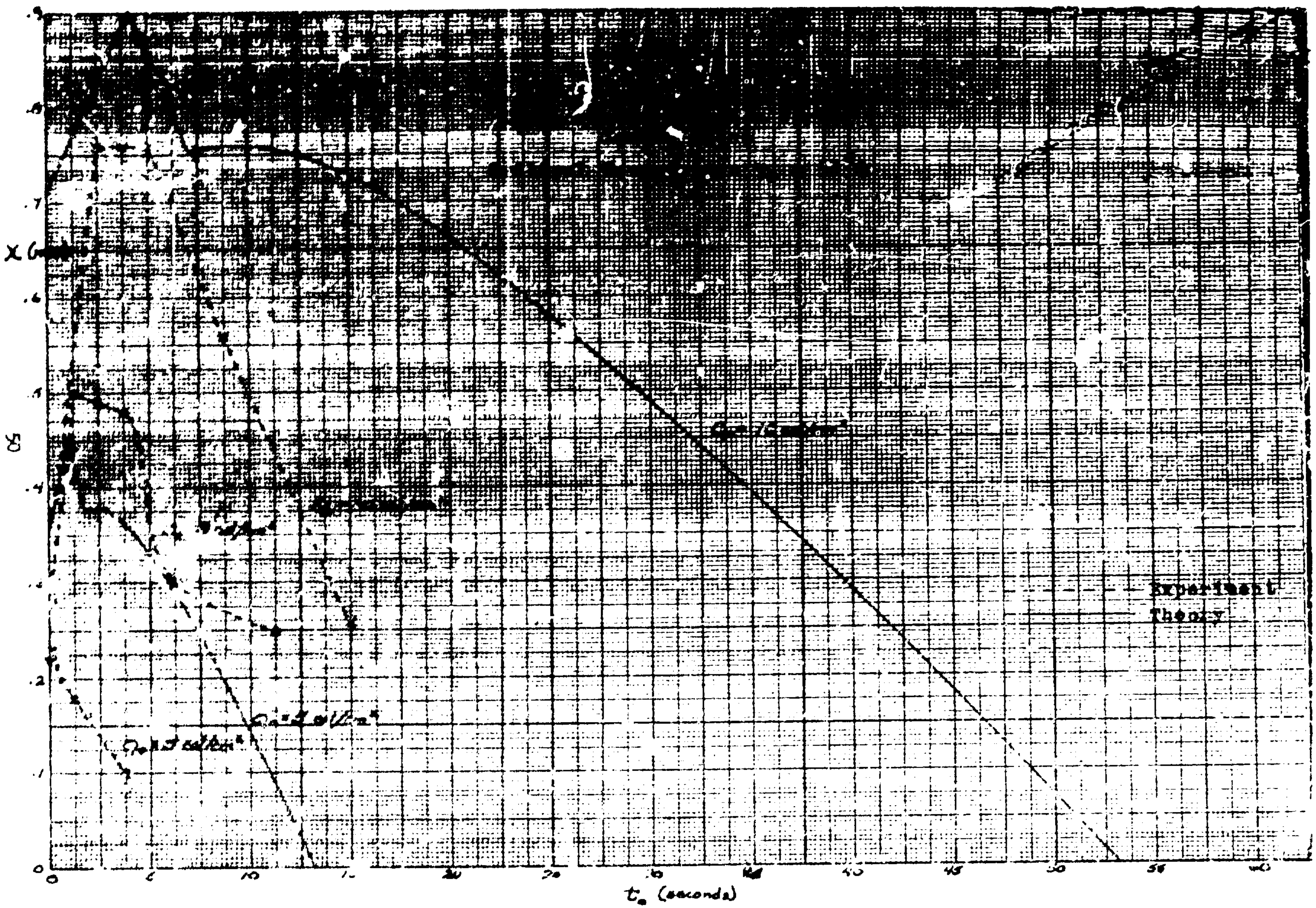
Now, we noted earlier that the skin does not react like an opaque object. The fact that this is so reduces the temperature for the deep dermal burn. Since this is so, we know that our calculated depths of injury are too high. We can compensate for this by raising T_c to some effective value higher than that obtained from the data of Henriques and Alpen. The effect of shifting T_c to 58°C* is shown in Figures 6 and 7. The change in depth of damage is not excessive. In other words, the flash burn damage depth is not excessively critical to the fact that the skin is translucent.

It is now of interest to compare our calculations with actual experimental square pulse data. The dotted curves on Figures 4, 5, 6 and 7 are experimental points taken from Figure 2 of Reference 5. The reader should note that the accuracy of the experimental data cannot be very good. In fact, the experimental depth of damage as determined from measured depth of collagen damage (Figure 2, Ref. 5) does not agree at all well with the same data expressed in quarters of

* 58°C was chosen to obtain a rough fit to the shape of some experimental curves given in Reference 5.




X
0.0
.2
.4
.6
.8
1.0
1.2
1.4
20
40
60
80
100
120
140
160
180
200
220
240
t_o (seconds)
Experiment
Theory


X
.9
.8
.7
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.3
.2
.1
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0
5
10
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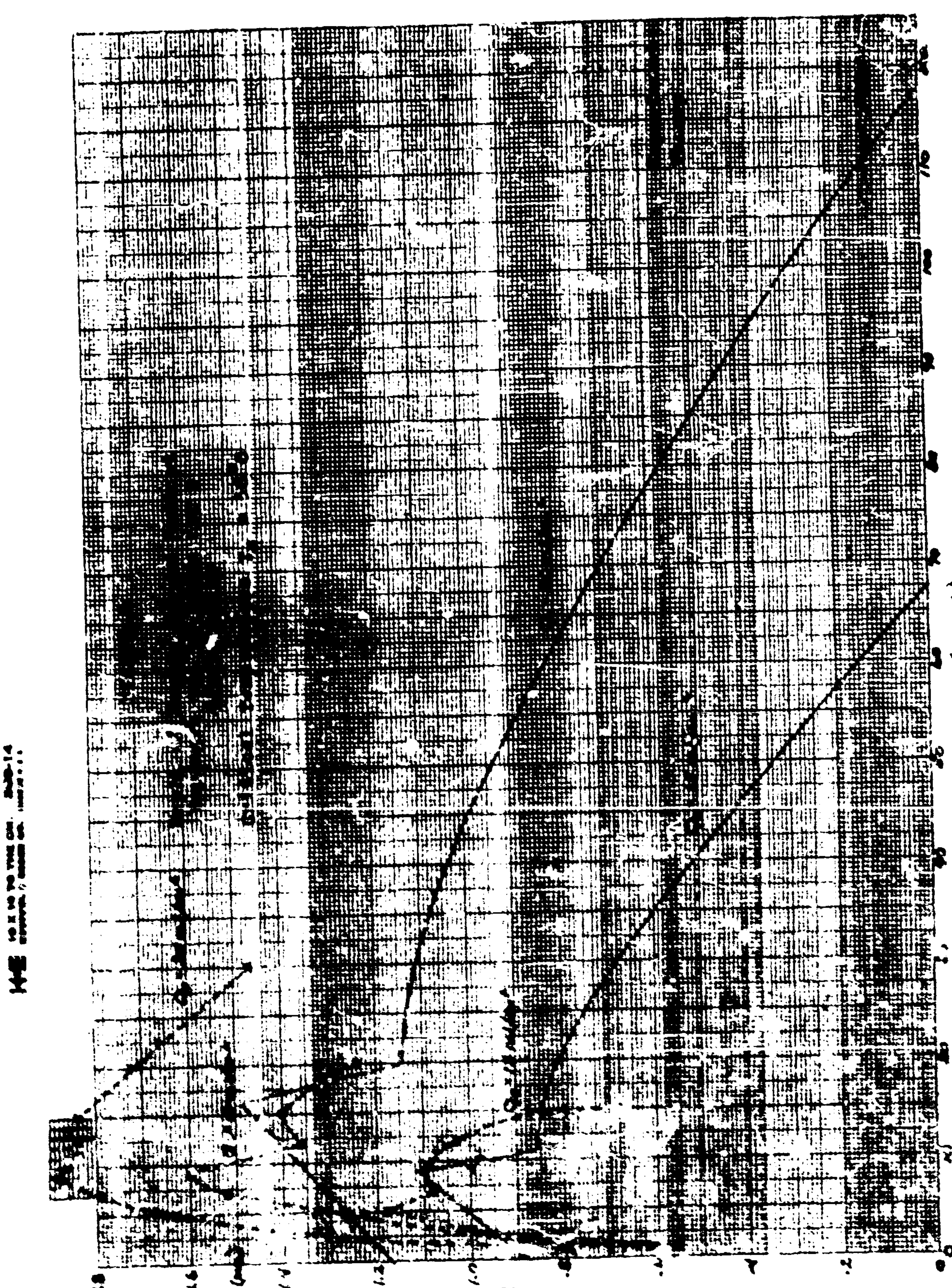
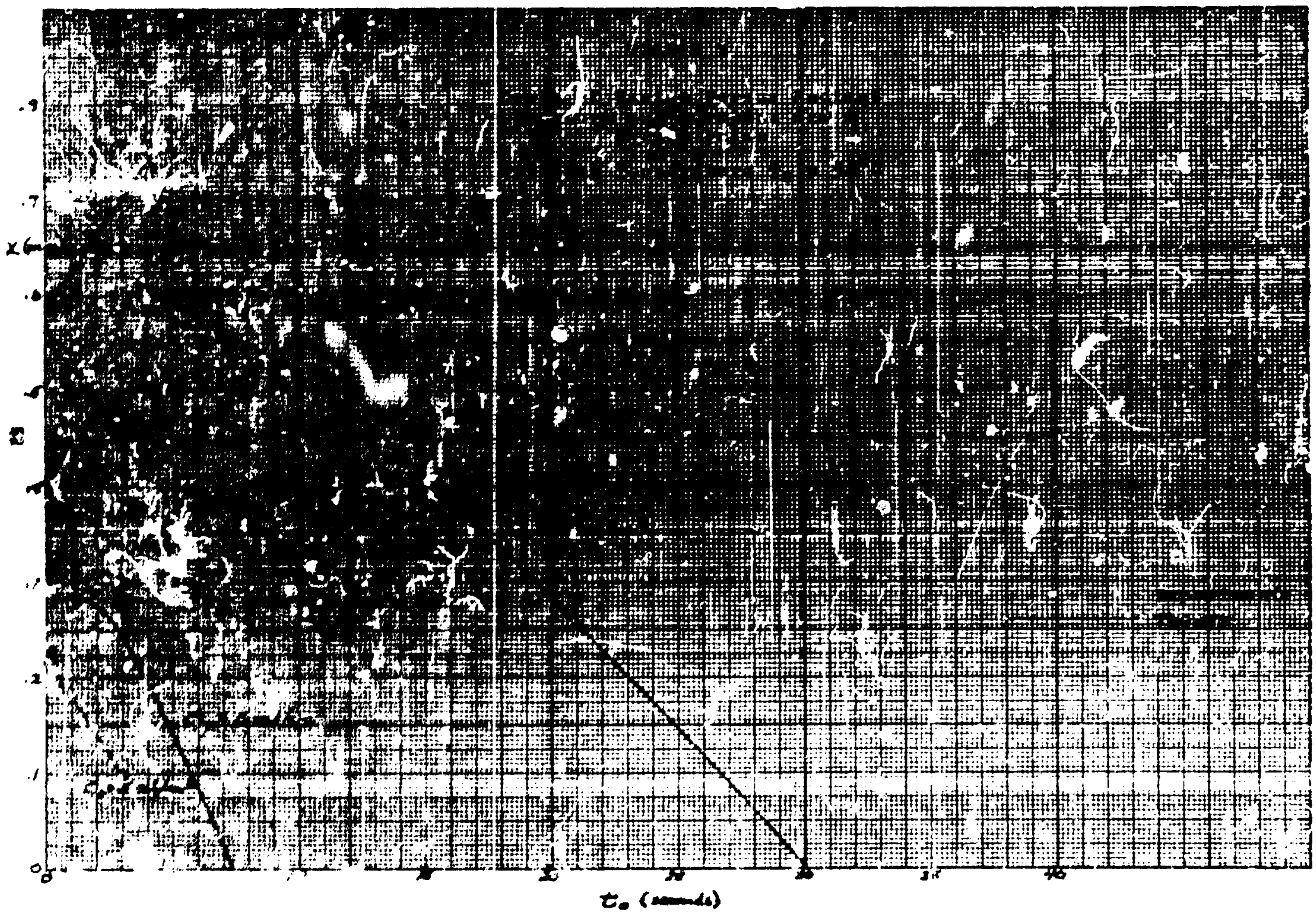
25
30
35
40
45
50
55
60
t_o (seconds)
Experiment
Theory


1.8
1.6
1.4
1.2
1.0
.8
.6
.4
.2
0
10
20
30
40
50
60
70
80
90
100
110
120
τ₀ (seconds)



.9
.7
.6
.5
.4
.2
.1
0
10
20
30
40
t_o (seconds)

dermal collagen destroyed (Figure 3, Ref. 5). The error in time at which maximum damage occurs is also large (see Table 1, Ref. 5). The times vary by factors of 2 and 3. Thus, we have quite reasonable quantitative agreement with the experimental data. We cannot expect good agreement between the shapes of the experimental and theoretical curves for the reasons indicated above. The comparison of theory and experiment at the maximum looks as follows for the case $T_c = 58^\circ C$.

Table 2

Theoretical and Experimental Depth of Maximum Collagen Damage for Various Values of Q_o

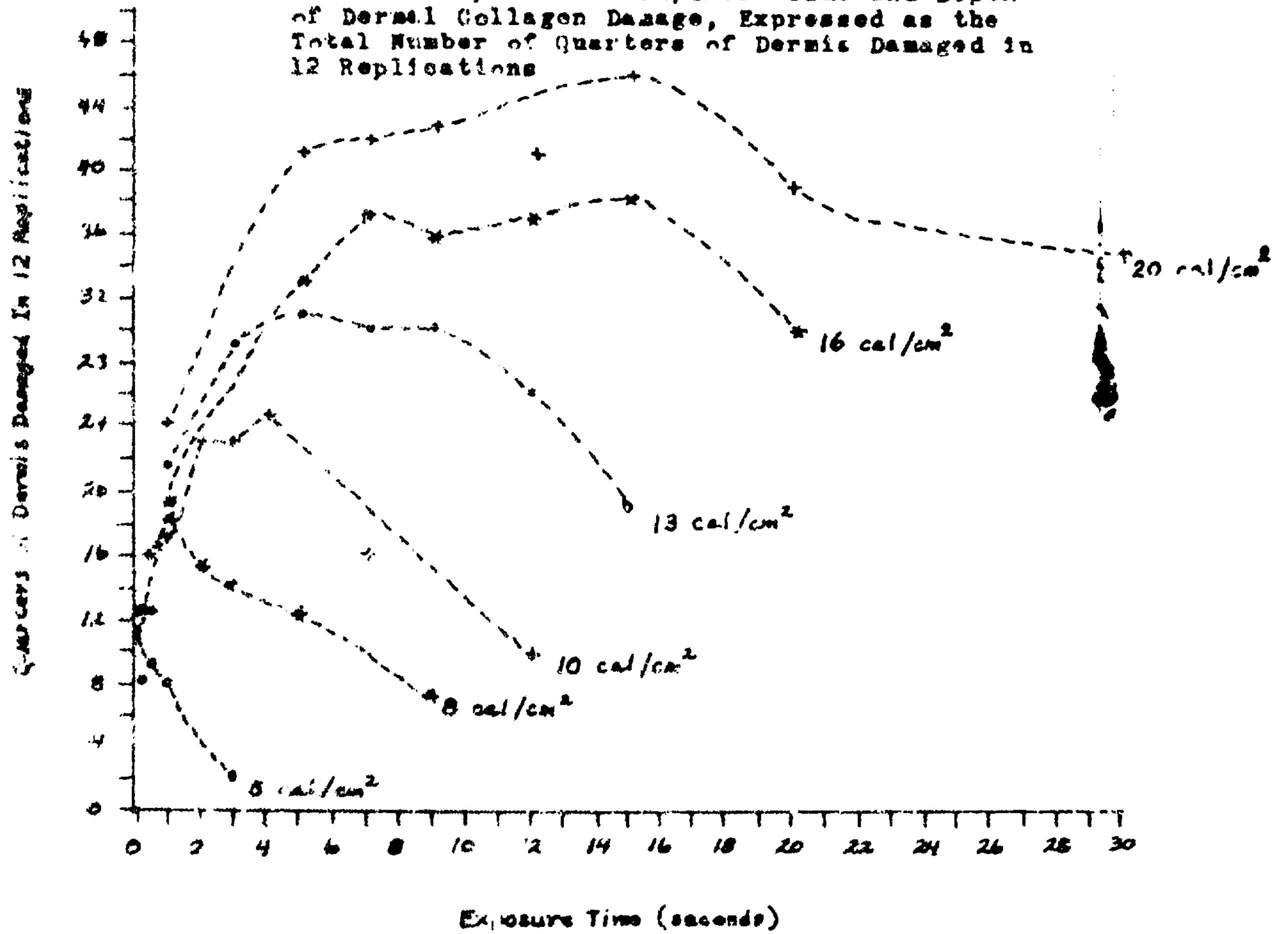
Q_o (cal/cm^2)	Experimental x_m(mm)	Theoretical x_m(mm)
20	1.9	1.35
16	1.6	
15		1.0
13	1.2	
10	.8	.7
8	.5	
5	.2	.3

In making the comparison we have taken one-half the boiling point jump as a reasonable compensation for the fact that the transition to a new temperature distribution cannot occur immediately.

To show the variability in the experimental data, we have reproduced Figure 3 of Ref. 5 in Figure 8 of this chapter and Figure 2 of Ref. 5 as Figure 11 of this chapter. The reader should note the large change in the shape of the experimental curves when the injury is expressed in quarters of dermis destroyed. The maximum at 20 cal/cm^2 comes at 16 seconds in Figure 3 of the reference whereas it comes at about 8 seconds in Figure 2 of the reference. Thus, the shapes of the experimental curves cannot be very accurate due to the inherent difficulties in estimating damage depth in edematous tissue. The curves in Figure 3, however, agree rather well with the [illegible] theoretical shape. One can see the rather flat behavior of the 20 cal/cm^2 curve to the right of the

FIGURE 8

Relationship Between Exposure Time and Depth of Dermal Collagen Damage, Expressed as the Total Number of Quarters of Dermis Damaged in 12 Replications



maximum and there is even evidence of readjustment of the temperature distribution as boiling occurs at about 20 seconds. One also sees the linear decline to the left of the maximum with a sharp break downwards presumably when partial steam blow-off occurs. We have not attempted to estimate the point of steam blow-off theoretically.

We can say, I believe, with considerable reliability that heat given in a very short time with steam blow-out will give the surface appearance of a 3rd degree (full skin thickness) burn whereas this will not be the case at all. Thus, one cannot expect correlation between surface appearance and depth of injury, particularly in extreme cases.

Finally, as to the time at which the theoretical maximum damage is reached, we can only say that this occurs at approximately the boiling point and perhaps slightly earlier. At the boiling point, our equation (10) yields the result

$$Q_o = 4.96\sqrt{t_o}$$

This approximate relation between total calories per cm^2 and pulse duration at the maximum is plotted in Figure 9. The measured relationship from Reference 5 is also shown. The agreement is rather startling. One notes right away that the measured curve of Log Q_o versus t_o has slope approximately 1/2 as it should theoretically and also that theory and experiment do not differ appreciably.

As a further check on the theory we use the simulated bomb pulse data of Reference 6. Tables from this reference are given below.

Table 3

Median Values at 5 cal/cm^2 Radiant Exposure

Exposure Time (sec)	Weapon Yield (KT)	Surface Appearance	Depth of Dermal Damage (mm)
1.8	20	[illegible]+M	0.055
2.5	40	2 [illegible]	0.00
4.0	100	2+[illegible]	0.00
12.6	1000	1+M	0.00

FIGURE 9

Relationship Between The Critical Exposure Time
For Maximal Dermal Collagen Damage
Measured in Millimeters and Radiant Exposure

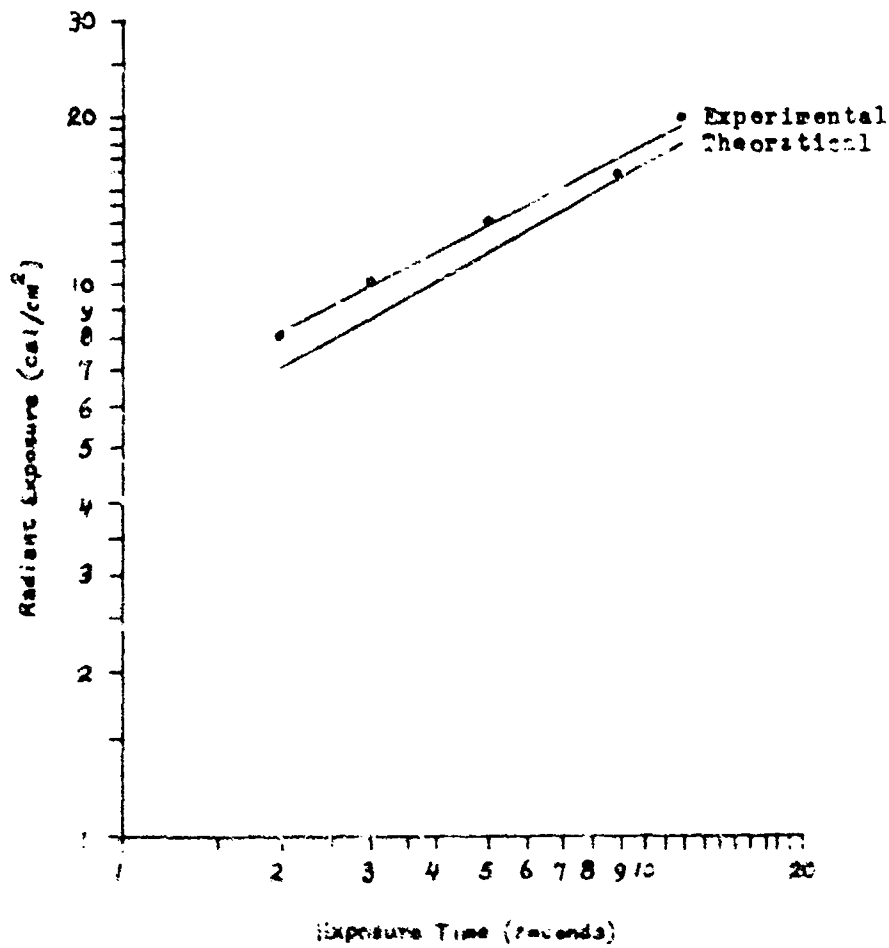


Table 4

Median Values at 10 cal/cm^2
Radiant Exposure

Exposure Time (sec)	Weapon Yield (KT)	Surface Appearance	Depth of Dermal Damage (mm)
1.8	20	3+M	0.46
2.5	40	3+S	0.35
4.0	100	3+M	0.46
12.6	1000	3+m	[illegible]
40.0	10000	2+S	0.07

Table 5

Median Values at 15 cal/cm^2
Radiant Exposure

Exposure Time (Sec)	Weapon Yield (KT)	Surface Appearance	Depth of Dermal Damage (mm)
4.0	100	3+S	0.92
12.6	1000	3+S	0.85
40.0	10000	3+S	0.62

Table 6

Median Values at 20 cal/cm^2
Radiant Exposure

Exposure Time (Sec)	Weapon Yield (KT)	Surface Appearance	Depth of Dermal Damage (mm)
4.0	100	5+m	[illegible]
12.6	1000	3+S	1.39
40.0	10000	3+S	1.43

Note:

1+ - erythema	m - mild
2+ - patchy white burn	M - moderate
3+ - uniform white burn	S - severe
4+ - steam bleb	
5+ - carbonization	

In reading the tables the reader should note that the depths given refer to the dermis only. To get the total depth of damage one must add the epidermal thickness of about 0.1 mm. The comparison of tables with theoretical values is given below. This theoretical check was made by using the equivalent square pulse calculations (s), with $t_m = t_o/7.85$. We shall present the same comparison for the theoretical bomb pulse.

Table 7

Comparison of Theoretical and Calculated Depths of Damage

Weapon Yield	$Q_o = 5$ Exp	$Q_o = 5$ Th	$Q_o = 10$ Exp	$Q_o = 10$ Th	$Q_o = 15$ Exp	$Q_o = 15$ Th	$Q_o = 20$ Exp	$Q_o = 20$ Th
20	.16	.28	.56	.63				
40	.14	.27	.45	.64				
100	.12	.26	.56	.68	1.02	1.04	1.18	[illegible]
1000	.05	0	.41	.54	.95	1.08	1.39	1.52
10000			.17	.15	.72	.75	1.43	1.15

The agreement between theoretical and experimental points is well within the variability of the experimental data.

The reader should note that for the case $Q_o = 5$, the mean value of dermal damage was used rather than the median value as given in Table 3. In this case, the mean and the median differed significantly. This was not true in the other cases.

Now, we wish to show that calculations based on a square wave pulse of duration 7.85 t_m do not differ significantly from bomb pulse calculations of the same total energy. The bomb pulse calculations are given in the table below for $T_c = 58°C$ and $T_c = 52°C$. The former value fits the data better as we have noted earlier.

The results in Table 8 may be compared with Table 7, using $T_c = 58°C$. Agreement is excellent except in a few cases where the weapon yield is high and the depth of damage small.

Finally, we show in Figure 10 the two lines bounding the dermal burn area. The top line is, of course, the line along which maximum depth of burn occurs as plotted in Figure 8. The lower line is the line on which the critical temperature is just reached at the surface of the skin.

FIGURE 10

Q_o Versus Yield for the Lines Bounding the Burn Region

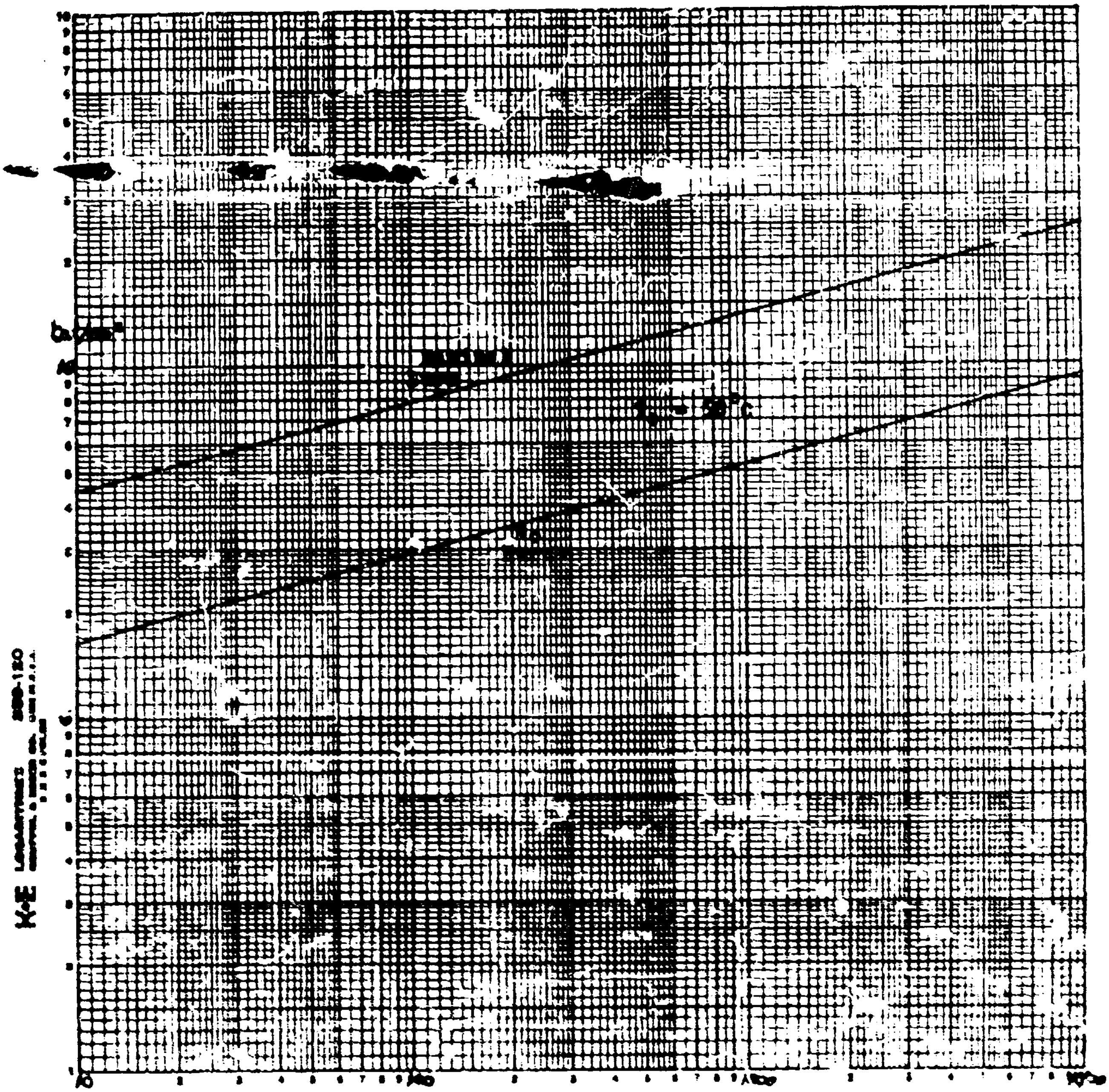


FIGURE 11

Relationship Between Exposure Time and Average Depth of Damaged Collagen for Each Radiant Exposure.

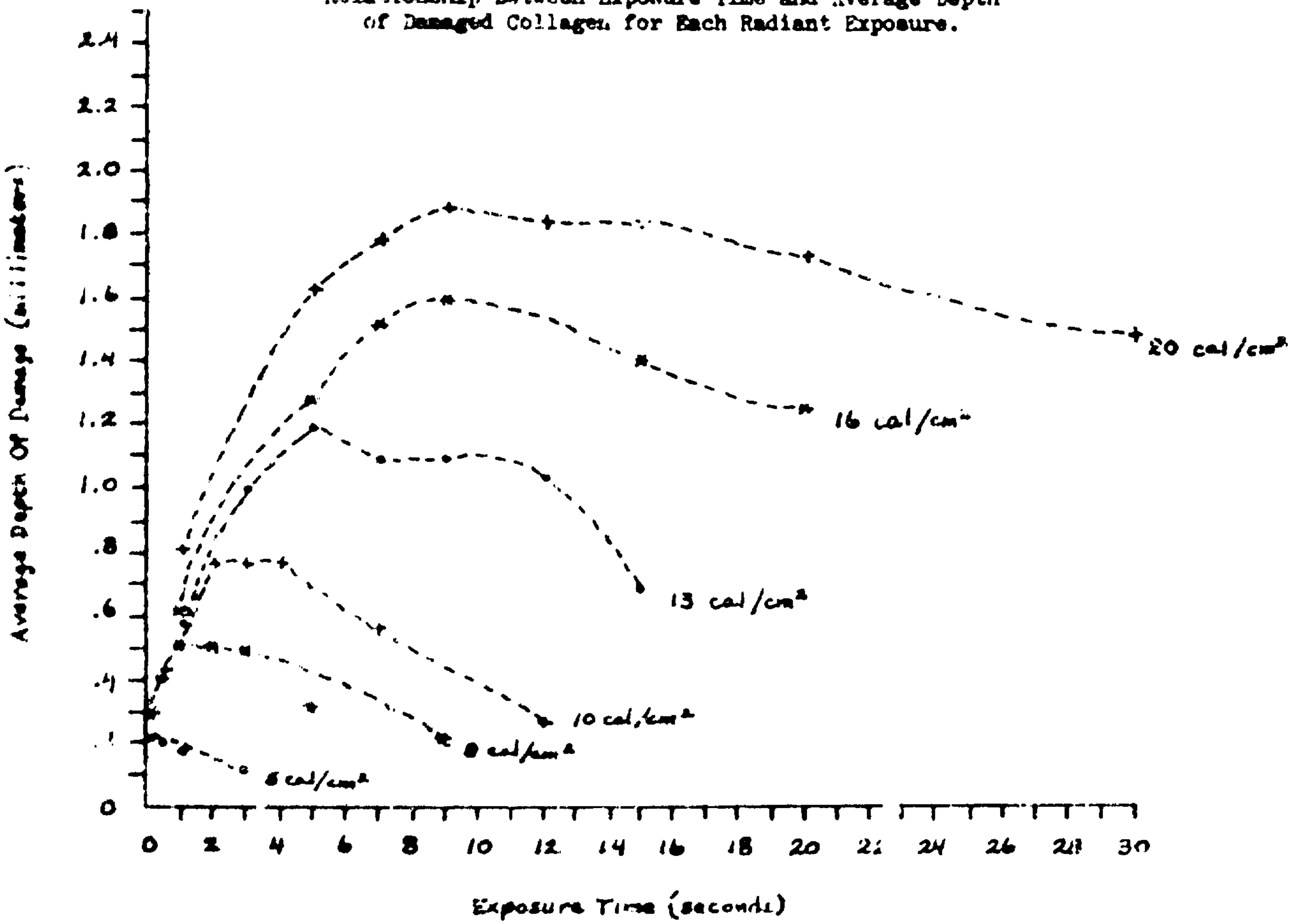


Table 8

Depth of Damage Versus Bomb Yield For The Thermal Pulse of a Weapon

$T_c = 52^\circ C$				$T_c = 58^\circ C$			
W	Q_o		x_m (mm)	W	Q_o		x_m (mm)
1	20	B	1.45	1	20	B	1.20
10	20	B	1.48	10	20	B	1.22
20	20	B	1.49	20	20	B	1.23
40	20	B	1.51	40	20	B	1.25
100	20	B	1.53	100	20	B	1.26
1000	20	B	1.62	1000	20	B	1.34
10000	20	NB	1.53	10000	20	NB	.980
1	15	B	1.10	1	15	B	.910
10	15	B	1.12	10	15	B	.926
20	15	B	1.14	20	15	B	.943
40	15	B	1.15	40	15	B	.951
100	15	B	1.17	100	15	B	.967
1000	15	B	1.25	1000	15	B	1.03
10000	15	NB	.980	10000	15	NB	.55
1	10	B	.743	1	10	B	.614
10	10	B	.772	10	10	B	.638
20	10	B	.783	20	10	B	.647
40	10	B	.799	40	10	B	.661
100	10	B	.822	100	10	B	.680
1000	10	NB	.730	1000	10	NB	.440
10000	10	NB	.400	10000	10	NB	.070
1	5	B	.390	1	5	B	.322
10	5	B	.417	10	5	B	.345
20	5	NB	.400	20	5	NB	.265
40	5	NB	.375	40	5	NB	.240
100	5	NB	.330	100	5	NB	.200
1000	5	NB	.145	1000	5	NB	0
10000	5	NB	0	10000	5	NB	0

Note: B - boiling
NB - not boiling

CONCLUSIONS

1. The theory presented here is adequate to predict depth of damage from thermal pulses.

2. Depth of burn can probably not be assessed clinically with accuracy by surface appearance.

3. A square pulse of duration $t_o = 7.85\, t_m$ and height $1/7.85\, (dQ/dt)_{max}$ is an adequate simulation of a bomb pulse, where $(dQ/dt)_{max}$ is the maximum intensity in the bomb pulse.

4. Tissue necrosis occurs at a critical temperature $T_c = 52^{\circ}C$ for heat application of the duration range of bomb pulses.

5. The 2° burn curve in TM 23-200 should be dispensed with and actual depth of damage versus Q_o and duration should be used.

Computation of Partial Pulse Data

We wish to consider the case where personnel are exposed to a given fraction of the bomb thermal pulse and to compute the associated depth of damage. From a tactical standpoint, exposure to a partial pulse occurs, of course, when personnel are able to take evasive action and reach a region of partial or total thermal shielding. Evasion is possible only for the larger bombs, the lower limit of bomb size being in excess of 100 KT.

From a mathematical standpoint, we use the expression (3) for the bomb pulse, with the further condition that $dQ/dt = 0$ after some critical time $t = t_c$. Thus one has

$$\frac{dQ}{dt} = \frac{Q_c}{4\sqrt{\pi}} K^3 t^{-5/2} \epsilon^{-K^2/4t} \qquad 0 \le t \le t_c \tag{23}$$

$$\frac{dQ}{dt} = 0 \qquad t > t_c$$

It appeared to us, at first, that an exact solution to this problem was not possible since the required LaPlace transforms were not tabulated and appeared impossible to evaluate in any convenient analytical form. However, it was noticed that the problem, stated in the form of a Green's function integral, could be solved in closed form. Thus, one formulates the equivalent solution as follows:

At $t = t_c$, the temperature distribution is given by (5), i.e.,

$$T(x,t_c) = \frac{\epsilon Q_c}{k a \sqrt{\pi t_c}} \left[\frac{K(K+ax)}{2t_c} + 1 \right] \epsilon^{-\frac{(K+ax)^2}{4t_c}} \tag{24}$$

We then seek a function $T_+(x,t)$ such that

$$T_+(x,0) = T(x,t_c)$$

and

$$\left.\frac{\partial T_+}{\partial x}\right|_{x=0} = 0 \qquad t > 0$$

The problem is then solvable in terms of a Green's function integral with kernel $G(x,x',t)$ such that

(1) G satisfies the heat flow equation, and

(2) G satisfies the boundary condition

$$\left.\frac{\partial G}{\partial x}\right|_{x=0} = 0 \qquad \text{for all} \qquad t > 0$$

The function $G(x,x',t)$ turns out to be

$$G(x,x',t) = \frac{a}{2\sqrt{\pi t}}\left[\epsilon^{-\frac{a^2(x-x')^2}{4t}} + \epsilon^{-\frac{a^2(x+x')^2}{4t}}\right] \tag{25}$$

and the required solution for $T_+(x,t)$ is

$$T_+(x,t) = \frac{a}{2\sqrt{\pi t}}\int_0^\infty\left[\epsilon^{-\frac{a^2(x-x')^2}{4t}} + \epsilon^{-\frac{a^2(x+x')^2}{4t}}\right]T(x',T_c)\,dx' \tag{26}$$

If one introduces the notation

$$x = \frac{K}{a}u\,; \quad x' = \frac{K}{a}u'\,; \quad \lambda^2 = \frac{K^2}{4t}\,; \quad \lambda_c^2 = \frac{K^2}{4t_c}$$

the integral (26) reduces to

$$T_+(u,\lambda,\lambda_c) = \frac{\epsilon Q_0 K}{2\pi\sqrt{t\,t_c}\,ka}\int_0^\infty\left[\epsilon^{-\lambda^2(u-u')^2} + \epsilon^{-\lambda^2(u+u')^2}\right]\left\{2\lambda_c^2(1+u')+1\right\}\epsilon^{-\lambda_c^2(1+u')^2}\,du' \tag{27}$$

Then, setting

$$p(t,t_c) = \lambda^2+\lambda_c^2$$

$$q(t,t_c,u) = \lambda_c^2 - \lambda^2 u$$

$$q_+(t,t_c,u) = \lambda_c^2 + \lambda^2 u$$

$$r(t,t_c,u) = \lambda_c^2 + \lambda^2 u^2$$

One evaluates (27) and finds

$$\begin{aligned}T_+(u,\lambda,\lambda_c) = \frac{\epsilon Q_0 K}{2\pi\sqrt{p\,t\,t_c}}\,\frac{\epsilon^{-r}}{ka}\Bigg\{&\left[\frac{\sqrt{\pi}}{2}\left(1+2\lambda_c^2\left(1-\frac{q}{p}\right)\right)\mathrm{erfc}\,\frac{q}{\sqrt{p}} + \frac{\lambda_c^2}{\sqrt{p}}\,\epsilon^{-q^2/p}\right]\epsilon^{q^2/p}\\ &+\left[\frac{\sqrt{\pi}}{2}\left(1+2\lambda_c^2\left(1-\frac{q_+}{p}\right)\right)\mathrm{erfc}\,\frac{q_+}{\sqrt{p}} + \frac{\lambda_c^2}{\sqrt{p}}\,\epsilon^{-q_+^2/p}\right]\epsilon^{q_+^2/p}\Bigg\}\end{aligned} \tag{28}$$

Equation (28) is the exact solution of the problem as one may verify directly.

We must now consider the limitations imposed on the calculation by the occurrence of boiling at the surface. Our viewpoint will be, insofar as the partial pulse is concerned, that evasion is ineffective if burns which result in boiling conditions cannot be avoided. In any event, we know that the depth of burn is sharply attenuated when boiling occurs and hence we shall limit partial pulse calculations to sub-boiling conditions. Thus, we impose the condition

$$(29) \qquad T(0,t_c) \le T_o$$

where $T(0,T_c)$ is given by (24). The condition (29) also limits the values of x which we must consider. For injury cannot occur unless $T_+(x,t)$ reaches the critical temperature T_c. Thus, the depth x is limited by the condition

$$(30) \qquad T_+(x,t) \ge T_c$$

or, put another way,

$$(31) \qquad \frac{T_+(x,t)}{T_o} \ge \frac{T_c}{T_o} = .35$$

for an effective critical temperature of 58°C. If the critical temperature is set at 52°C, the condition is

$$(32) \qquad \frac{T_+(x,t)}{T_o} \ge .26$$

The latter condition is sufficiently broad to cover any reasonable value of critical temperature.

Calculations of depth of injury for partial pulses are given in the table below. The bomb sizes considered are 1 MT and 10 MT with critical evasion times of 1 second and 2 seconds.

PARTIAL PULSE CALCULATIONS

Case w = 1000 KT

x (cm)	t_0 (sec)	$\frac{T_{max}}{\epsilon Q_0}$
0	≥ 2.07	11.50
.02	≥ 3.19	8.36
.04	≥ 4.63	6.43
0	2.00	11.58
.02	2.00	7.65
.04	2.00	4.67
0	1.00	8.07
.02	1.00	4.44
.04	1.00	1.73

Case w = 10000 KT

x (cm)	t_0 (sec)	$\frac{T_{max}}{\epsilon Q_0}$
0	≥ 6.63	6.52
.02	≥ 8.55	5.35
.04	≥ 10.8	4.51
0	2.00	1.31
.02	2.00	.982
.04	2.00	.463
0	1.00	.880
.02	1.00	.176
.04	1.00	.148

APPENDIX A

APPENDIX A

It is realized that some of the readers of this report will not be familiar with all of the mathematical techniques for solving the heat flow equation. The solutions can always be checked, however, by the following process. First, the solution must satisfy the heat flow equation

$$(1) \qquad \frac{\partial^2 T}{\partial x^2} = \frac{\rho c}{k} \frac{\partial T}{\partial t}$$

where we have set $\rho c/k = a^2$ in the text.

Second, the solution must satisfy the required initial condition and the required boundary conditions. Thus, for example, the solution (5) on page 35 must satisfy the initial condition $T(x,0) = 0$ at $t = 0$ for all x since we have defined T as the excess of the skin temperature over its initial temperature. It is clear that since the exponential term in (5) approaches zero for all x as $t \to 0$ that the required initial condition is satisfied.

Now, also in the case of (5), the boundary condition at the surface is that the radiant heat input $\epsilon \frac{dQ}{dt}$ must at all times equal the heat conducted away from the surface $-k \left.\frac{\partial T}{\partial x}\right|_{x=0}$. Thus, one computes $-k \left.\frac{\partial T}{\partial x}\right|_{x=0}$ from (5) and checks to see if it is equal to $\epsilon \frac{dQ}{dt}$ as given by (2). Finally, one may easily differentiate (5) to show that $T(x,t)$ satisfies the heat flow equation.

This same process of checking may be followed throughout the mathematics. For example in Eq. (26) on the partial pulse calculation, it is obvious that $T_+(x,t)$ satisfies the heat flow equation as one can quickly verify by differentiating under the integral sign. Further, since there is no heat input for

$t>0$, $\left.\frac{\partial T_+}{\partial x}\right|_{x=0} = 0$ for all $t > 0$ and it is easily shown that this is true since

$\left.\frac{\partial G}{\partial x}\right|_{x=0} = 0$ for all $t > 0$. Further, $T_+(x,t)$ must satisfy the initial condition that $T_+(x,0) = T(x,T_c)$. It is clear that this initial condition is satisfied, however, since

$$\epsilon^{\frac{-a^2(x+x')^2}{4t}} \rightarrow 0 \qquad \text{as } t \rightarrow 0 \text{ for all } x > 0$$

and

$$\epsilon^{\frac{-a^2(x-x')^2}{4t}} \longrightarrow 0 \qquad \text{as } t \rightarrow 0 \text{ for all } x > 0$$

unless $x = x'$. Thus, the $\lim\limits_{t \rightarrow 0} T_+(x,t) = T(x,T_c)$

Section 2. Time to Shock.

In this section we wish to compute the time from burn injury to onset of hypovolemic shock. There are certain limitations to the calculation which cannot be resolved. These are:

1. The rate of fluid loss depends, of course, on the particular area of the body which is injured. This is evidently a physical phenomenon in the sense that fluid losses are impeded by the build up of back pressure in the region of loss. The capability of the particular body region to build back pressure depends on the "looseness" of the skin. Thus, for example, fluid losses are higher in burns to the backs of the hands.

2. Fluid replacement needs are known only from general clinical observations. Thus, one has such general observations as the loss rate per unit area for a burn assessed clinically as 2^{o} and the observation that the rate is twice as much for a burn assessed clinically as 3^{o}. We also have the general observation that fluid loss rate is a linear function of total tissue destroyed for area burns up to 50% total body area. None of these observations are exact in a scientific sense.

3. The total fluid loss required for hypovolemic shock is not known accurately.

The limitations listed above are not as serious as one might think. First of all, the critical body areas where fluid loss is rapid coincide with areas which cause early incapacitation for reasons other than shock, i.e., the backs of the hands and the face. As to 2., it is true that the clinical assessments of burn area and depth versus fluid loss are not in the category of a physical measurement, but these assessments tend to be valid statistically since the burn surgeon ultimately finds, from required skin grafts, the approximate extent of the injury. The lack of knowledge in 3. is not serious enough to affect the calculations appreciably. We have used 35% loss of blood volume as a criterion for shock and raised this to 42% when the loss is corrected for plasma protein balance acquired through flow of interstitial fluid into the circulating blood. This criterion must be regarded as conservative on account of the correction for protein balance and also because of the probability that neurogenic shock can also occur or that as Dr. Vogel says, "there is undoubtedly a neurogenic factor involved".

With these limitations in mind, we define the "statistical" 2^{o} burn as a burn which extends to half the dermal thickness which we shall take to be 1 mm. For the statistical 2^{o} burn we know that the fluid replacement requirement, aside from fluids required to replace insensible losses, is 2.0 cc fluid per kilogram body weight multiplied by the % of the body area burned, for the first 24 hours. The requirement is half this for the second 24 hours. We also know that the fluid loss <u>rate</u> is very probably exponential due to the build up of back pressure in the injured area. In addition, we shall take the fluid loss rate to be proportional to the total tissue destroyed. Thus, we set

$$R = R_o \epsilon^{-\alpha t}$$

where R_o is the initial rate of loss and α is the decay constant. For the statistical 2^{o} burn we have

$$\int_0^{24} R_o \epsilon^{-\alpha t} dt = 2wf \times 10^{-3} \qquad \text{(liters)}$$

$$\int_{24}^{48} R_o \epsilon^{-\alpha t} dt = wf \times 10^{-3} \qquad \text{(liters)}$$

where w = weight of body in kg

f = % body area burned to a depth of 1 mm

From these equations we find

(1) $\alpha = .029$

and $\quad (2) \quad R_o = \dfrac{2\alpha w f \times 10^{-3}}{1-\epsilon^{-24\alpha}}$

Since the average body weight of a man is 70 kg and the average blood volume is 5.3 liters, our shock criterion for the statistical 2^o burn is

$$\int_0^T R_o \epsilon^{-\alpha t}\, dt = 5.3\,(0.42) = 2.2 \qquad \text{(liters)}$$

or

$$R_o(1-\epsilon^{-\alpha T}) = 2.2$$

or

$$\epsilon^{-\alpha T} = 1 - 2.2\alpha/R_o$$

For burns whose depth averages other than 1 mm, we replace R_o in (2) by

$$R_o \cdot \bar{x}$$

where $\bar{x}$ is the actual average burn depth in millimeters.

We can now construct a table of time to shock versus average depth $\bar{x}$ and % body area burned f.

Table 2.1

Time to Hypovolemic Shock Versus Average Burn Depth and Percent Body Area Burned

f \ $\bar{x}$	.1	.3	.5	.7	1.0	1.3	1.5	1.7	2.0	4.0
10					(53)	32.1	25.8	21.4	17.2	7.6
15				47.6	25.8	17.2	15.5	12.4	10.7	5.2
20			(53.1)	28.6	17.2	12.4	10.7	9.3	7.6	3.8
25			36.9	20.7	13.1	9.6	8.3	7.2	6.2	2.8
30		(71.0)	25.8	16.2	10.7	7.9	6.9	5.9	5.2	2.4
40		36.9	17.2	11.4	7.6	5.9	4.8	4.1	3.8	1.7
50		25.9	13.1	9.0	6.2	4.5	3.8	3.4	3.1	.3

$\bar{x}$ = average burn depth in mm

f = % body area burned

The encircled points represent no shock at all since the times are later than 48 hours.

Section 3. The Protection Afforded by Clothing

The problem of protection afforded by clothing is one of the most difficult problems in the area of thermal burns. Before proceeding with numerical analysis, therefore, it will be of interest to consider the problem in a qualitative way in order to understand the physical factors involved.

First of all, one can set certain limits on the problem which separate very serious burns from burns of a less serious nature. Thus, if the clothing is ignited throughout, it is clear that one can, in general expect very much more serious burns than if ignition does not occur at all or if it occurs only on the exterior of very thick protective clothing. Second, the transmissivity and reflectivity of light weight clothing plays an important role. For example, the usual white civilian shirt will transmit about 30% of the incident radiation, reflect 50% and absorb only 20%. Thus, the reflectance and transmittance generally play a more important role than the absorptance which ultimately leads to ignition. In the case of darker, heavier clothing, absorption is overwhelming compared to the transmittance, and the reflectance is, at best, 20% to 30% of the incident energy. This applies to the usual military summer khaki and green as well as to winter clothing.

In the case of military summer clothing, the clothing can either heat to the ignition point, giving a very serious burn indeed; or it can heat to some point under ignition, in which case the clothing radiates the absorbed energy to the body causing a burn which is about as serious and sometimes worse than the burn which would have occurred had the skin been bare. This last statement seems curious, but it is adequately supported by the data.

In the case of heavy, six-layer, winter clothing one encounters the most difficult problem of all. It is very probably a problem in which the physical factors are too complex to allow theoretical treatment. To be sure, the ignition conditions for the outer layer or layers can be found. However, one then has to consider whether the fire can be put out by rolling on the ground or whether it will be snuffed out by the blast wave. The latter contingency seems likely. The treatment of heat conduction through the various layers is hardly possible on account of the variable air spacing between layers. Thus, on this problem one can probably do no more than give the best possible empirical answer based on available data. The total energy input for burns under heavy clothing will, in any event, be very high (on the order of 100 cal/cm^2, Reference 7).

The important thing to notice is that, except for very heavy clothing, the various phenomena associated with burns under clothing can be understood rather well theoretically and can be checked adequately experimentally. The chief experimental limitation on laboratory experiments, in cases where the clothing bursts into flame, is that very small burn areas are used and the ignited clothing generally falls without touching the skin of the animal. This is hardly likely to be the case for ignition of clothing on the back of a prone man or, indeed, for ignition of the entire front or back of the clothing of a standing man. The experimental limitations on this score will become evident in the discussion and analysis to follow.

I. The Physical Properties of Various Types of Military Summer Clothing.

Physical data available on clothing are confined almost entirely to military materials except for some data on light white sheeting. Our problem will be to show that theoretical calculations can be applied rather accurately to materials for which data are available. Extension of the results to civilian clothing can then be made when and if data are taken.

The table below gives the pertinent data which we have collected on the physical properties of military clothing.

Table 3.1

Physical Data on Military Summer Clothing

Material	w (oz/yd^2)	ϵ_3	ϵ_2	ϵ_1	$E = 1 - \epsilon_2$
White cotton sheeting	3.2	.36	.51	[illegible]	.49
Impregnated cotton sateens (FR)					
Black	9.1	nil	.04	.96	.96
Medium gray	8.2	nil	.28	.72	.72
Green poplin	5.4	.03	.20	.77	.80
Green poplin (FR)	6.6	.03	.24	.73	.76
Khaki	5.4	.08	.35	.57	.65
Khaki (FR)	6.5	.06	.35	[illegible]	.65

ϵ_3 = Transmittivity
ϵ_2 = Reflectance
ϵ_1 = Absorptance
$E = 1 - \epsilon_2$ = Effective incident energy

It is clear from the table that the transmittance depends both on the color of the fabric and the weight. Thus, if there is unit effective energy incident on the white fabric, for example, and if the absorption coefficient is λ (yds^2/oz), then the transmitted energy will be $\epsilon^{-\lambda w} = .36/.49 = .74$ (we have removed the energy reflected, of course), and the absorption coefficient is $\lambda = .096$. Thus, if the cloth thickness were doubled, the transmission would still be $(.74)^2 \simeq .55$. Hence, the transmittance of white cloth is high compared to the darker fabrics. The white cloth, on the other hand, reflects half the incident energy and this quality is quite important. We shall refer to Table 3.1 later in the discussion of burns under non-flaming fabric. First, however, we wish to discuss the problem of ignition of fabrics.

2. Ignition of Fabrics.

As the basic differential equation for ignition, we set

3.1 $$\rho c \frac{dT}{dt} = \epsilon_1 \frac{dQ}{dt} - \sigma(1 - \epsilon_2)(T^4 - T_0^4)$$

Here we have treated the thin fabric as though it heats uniformly and loses heat during the heating cycle to the outside only. Neither assumption is strictly true but the approximation is good. Equation 3.1 may be integrated directly for the case of a square pulse i.e., for $dQ/dt = Q_o/t_o$. We have seen earlier that a square pulse of duration 7.85 t_m approximates the bomb pulse adequately. Thus, setting $dQ/dt = Q_o/t_o$ one has

$$\frac{dT}{dt} + \frac{\sigma(1-\epsilon_2)}{\rho c}(T^4 - T_o^4) - \frac{\epsilon_1}{\rho c}\frac{Q_o}{t_o} = 0$$

or

$$\frac{dT}{T_o^4 + \frac{\epsilon_1 Q_o}{t_o}\frac{1}{\sigma(1-\epsilon_2)} - T^4} = \frac{\sigma(1-\epsilon_2)}{\rho c}\,dt$$

If one sets $b^4 = T_o^4 + \epsilon_1 Q_o/\sigma t_o(1-\epsilon_2)$, this equation integrates to

3.2 $$\operatorname{Log}\left|\frac{1+T/b}{1-T/b}\right| + 2\tan^{-1}T/b = 4b^3\frac{\sigma(1-\epsilon_2)}{\rho c}t + \operatorname{Log}\left|\frac{1+T_o/b}{1-T_o/b}\right| + 2\tan^{-1}T_o/b$$

$$0 \le t \le t_o$$

Thus, at $t = 0$, $T = T_o$. The maximum temperature is reached at $t = t_o$; so that if T reaches or exceeds the critical temperature of ignition at t_o, the clothing will burst into flame. Clearly, the critical condition for ignition is given by that combination of total energy Q_c and duration t_o which allows T to just reach the critical temperature T_1 at $t = t_o$. Or, analytically

$$T(t_o) = T_1$$

where T_1 is the ignition temperature.

Now, if we set $x = T_1/b$ and $F(x) = \operatorname{Log}\left|1+x/1-x\right| + 2\tan^{-1}x$, then (34) can be written

3.3 $$x^3\left[F(x) - F(x_o)\right] = 4T_1^3\frac{\sigma(1-\epsilon_2)}{\rho c}t_o$$

at $T = T_i$ when $t = t_o$, where x_o is, of course, T_o/b. Thus, for a fixed fabric, the left hand side is a function of Q_o/t_o and the right hand side is a function of t_o.

Thus, given T_i for the fabric and the pulse duration t_o, the ignition energy is given directly by 3.3. For practical use one plots the left hand member as a function of x and reads off the value of x at which the left hand member reaches $4T_i^3 \frac{\sigma(1-\epsilon_1)}{\rho c} t_o$. This yields a quick way of computing ignition energies if the ignition temperature T_i is known.

One should note that we will have, in general, two cases to consider. The first case is the case of colored cloth where $\epsilon_3 \approx 0$ and $\epsilon_1 = \epsilon_2$. The second case is that of white cloth where ϵ_3 is relatively large. In this latter case, the burn process is dominated by the transmitted energy as modified by the reflectance. Thus, serious burns always occur prior to clothing ignition unless the white cloth is very thick. This latter case is not apt to occur in civilian populations. With military clothing, one would increase the reflectance not by using white cloth but by suitable impregnation with reflecting metal particles. Thus, in general, we shall set $\epsilon_1 = 1 - \epsilon_2$ except for normal civilian white clothes where the burn process will be computed using the transmitted energy plus the energy radiated by the heated fabric, if the latter should be appreciable.

3. Ignition Temperatures.

We have been able to find no direct data on the ignition temperature of clothing materials. However, we have proceeded first by inferring the ignition temperature of newspaper as given in Figure 12-1 of TM 23-200. Ordinary newspaper has a weight of 5.0×10^{-3} gm/cm^2. Using a nominal emissivity of $\epsilon = 0.75$ (the result is not critical to emissivity due to the radiative T^4 law) and the theory as presented above, we find the ignition temperature to be about 1840°F or 1280°K. We then measured the ignition temperature of cotton clothing using a soldering tip heated with an acetylene flame. This measurement cannot be considered accurate; but it showed that the ignition temperature was between 1500°F and 2000°F. Since we do not know the ignition temperatures accurately, we based some sample calculations on ignition temperatures of 1000°K, 1100°K and 1280°K. We have plotted $\frac{\epsilon_1 Q_0}{\epsilon_2 w^{1/2}}$ vs. $\frac{\epsilon_2 w^{1/2}}{\rho}$ for each of the three ignition temperatures mentioned. The calculation procedure is easy and hence other ignition temperatures may be used as they become available. An accurate knowledge of ignition temperatures for various materials would be quite useful. The calculations given in the next section are, therefore, illustrative of final results and will represent an accurate picture for cotton clothing only to the extent that our estimate of ignition temperature is correct.

4. A Sample Calculation of Ignition Energy.

Referring to Eq. 3.1 , one can note that for the very small bombs where the energy input rate is very high for a given Q_0, the ignition condition will be given roughly by

$$\rho c \Delta T = (1 - \epsilon_2) Q_0$$

where ΔT is the rise in temperature above ambient. The above equation will be roughly correct since the time during which the radiative T^4 term can act is small. On the other end of the scale, with very large bombs, the heat input rate for the same Q_0 is low and the temperature creeps up very slowly as temperatures near the ignition point are reached. In other words, the radiative cooling terms have a long time to act and thus become predominant. The conclusion from the above observations is that there will be no simple relation between yield and ignition energy. The relation will depend critically on ignition temperature and somewhat less critically on ρ, c and ϵ_2. But, in any event, there will

FIGURE 5.1

$\frac{\epsilon_1 Q_{s}}{\epsilon_a \sqrt{W}}$ vs. $\frac{\epsilon_x \sqrt{W}}{\rho}$ FOR AN IGNITION TEMPERATURE $\varphi_0 = 1000°K$

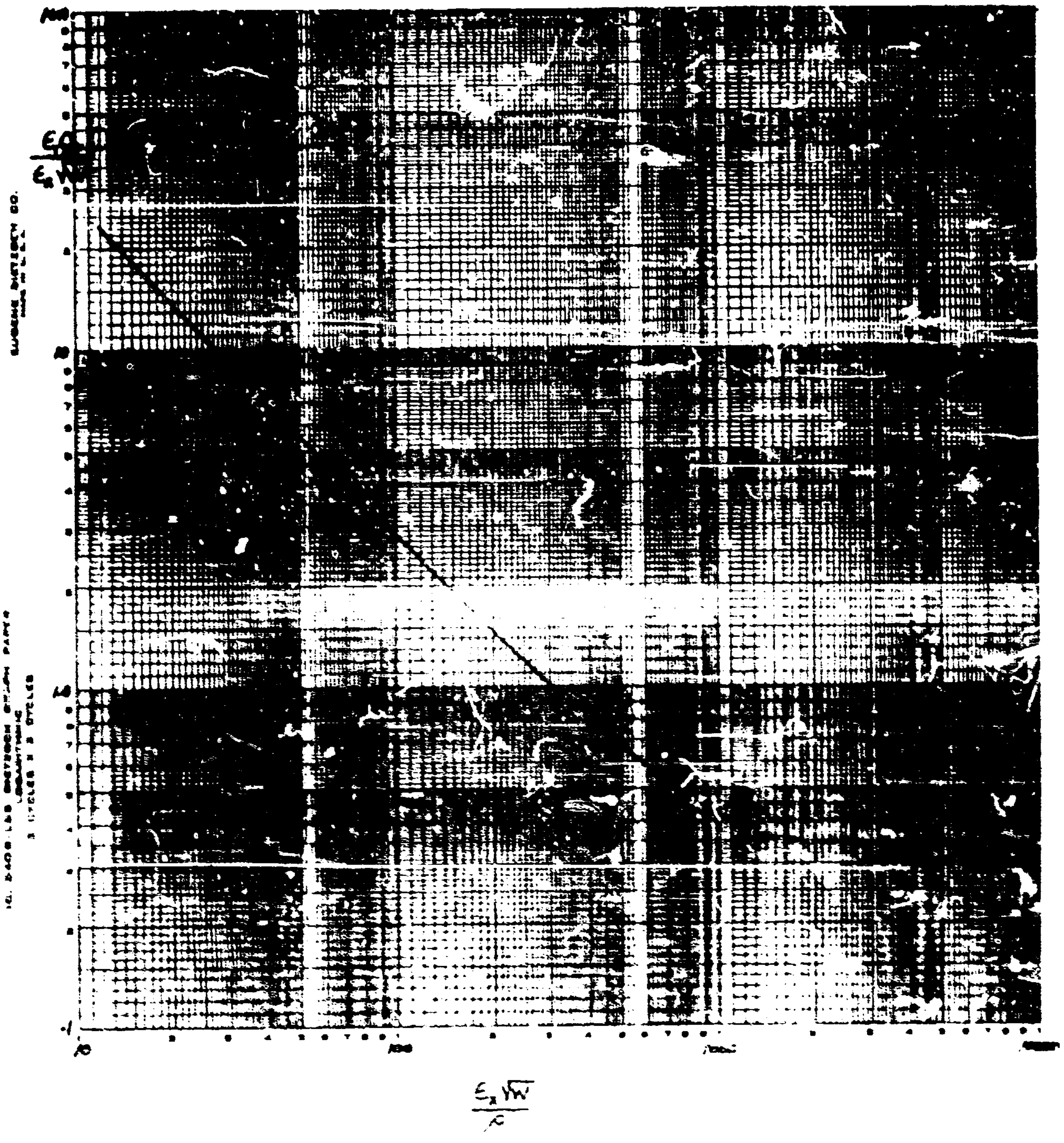
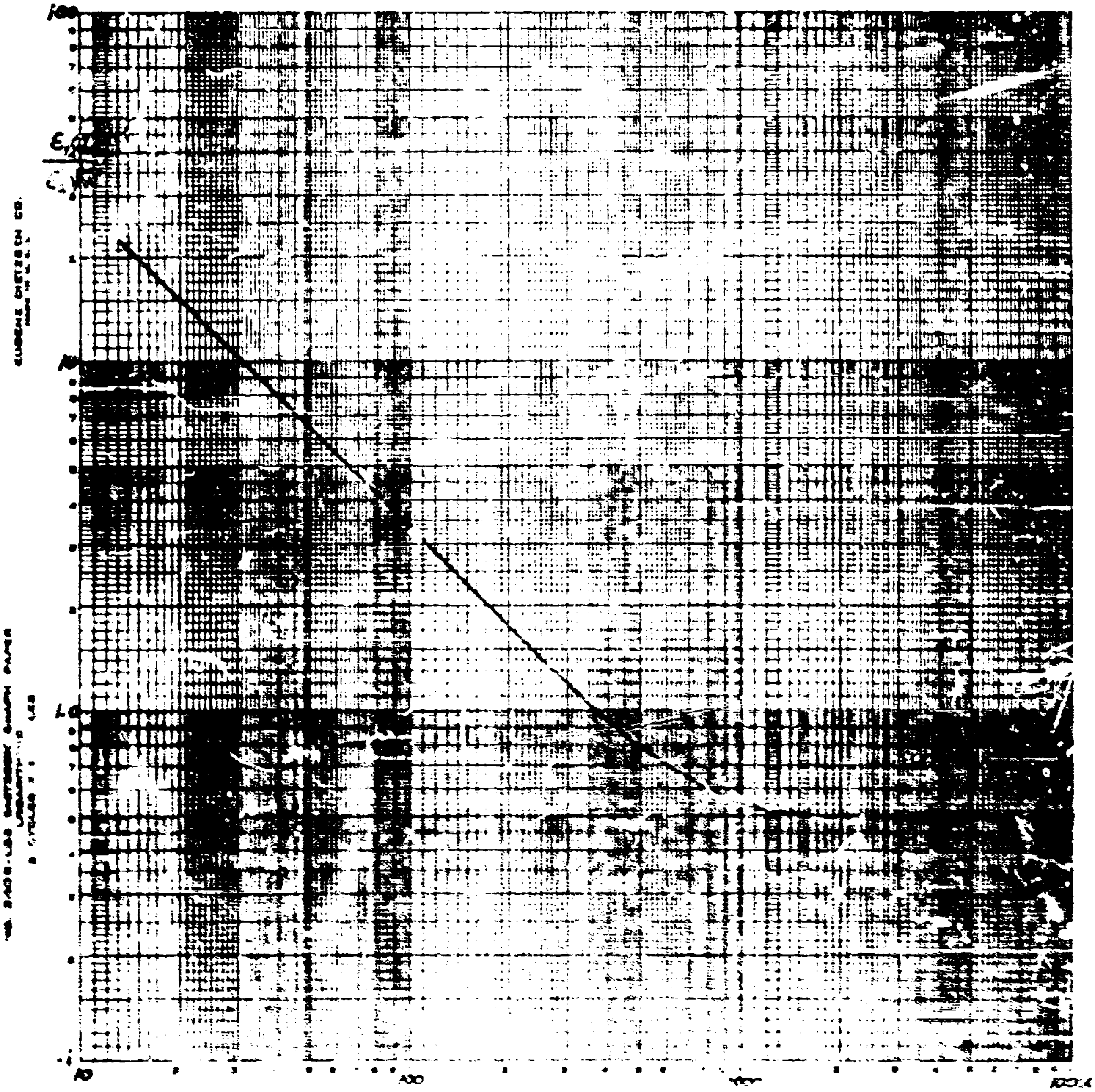


FIGURE 5.2

$\frac{\epsilon_1 Q_e}{\epsilon_2 \sqrt{W}}$ VS. $\frac{\epsilon_2 \sqrt{W}}{\rho}$ FOR AN IGNITION TEMPERATURE T_c = [illegible]°F

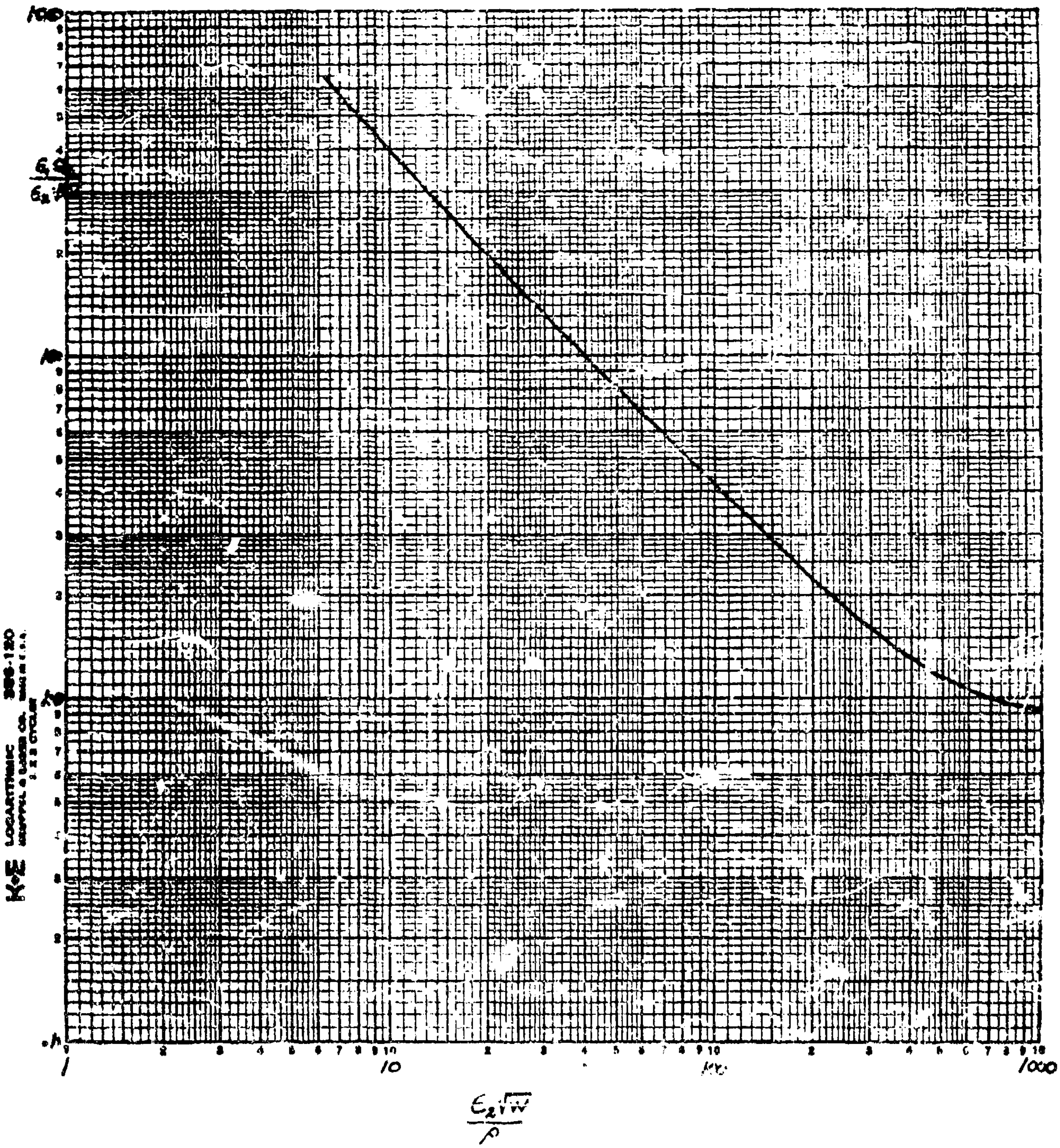


$\frac{\epsilon_2 \sqrt{W}}{\rho}$

H

FIGURE 5.3

$\frac{\epsilon_1 Q_0}{\epsilon_2 \sqrt{W}}$ VS. $\frac{\epsilon_2 \sqrt{W}}{\rho}$ FOR AN IGNITION TEMPERATURE $T_c = 1280^\circ K$



be no simple scaling law in terms of the yield w.

As an example of calculation of ignition energy we shall use one type of relatively heavy khaki having the following physical constants:

$$\rho = 16 \times 10^{-3} \ gr/cm^2$$

$$c = 0.40$$

$$1 - \epsilon_2 = .35$$

$$\epsilon_3 = 0$$

The heat capacity c is an estimated average value for heavy molecules in the given temperature range. This estimate was taken from all available data from the Bureau of Standards and can be regarded to be as accurate as necessary for calculation purposes. The ignition temperature T_i generally is the more critical parameter. In the calculation below, T_i was given the high value of 1840°F.

Table 3.2

Ignition Energies for Cotton Khaki Fabric, Weight 12 oz/yd^2

w (kt)	Q_o (cal/cm^2)
1	24
10	24
100	28
1000	36
10000	93

The sharp rise in Q_o with bomb size is characteristic of the effect of the T^4 radiation cooling. One can see from Figures 3.1, 3.2 and 3.3, the extent of the sharp rise is very critical to ignition temperature.

5. Burns Under Summer Clothing for Cases Where the Clothing Does Not Ignite.

We shall consider the case of ordinary military clothing (not white) where the cloth is essentially opaque to the incident energy. Since the skin temperature can never exceed the boiling point except under very extreme conditions of heating, the clothing will radiate both to the outside and to the skin approximately as though the temperatures on each side were $0^{\circ}K$, i.e., burns will be unimportant for maximum clothing temperatures below $800^{\circ}K$: ambient temperature will be of the order of $300^{\circ}K$ and the skin boiling temperature $373^{\circ}K$. Thus, any temperature correction for a -radiation to the cloth will be of the order of $(373/800)^4 \approx .05$; a correction of 5% is not warranted by the accuracy of other physical parameters. Radiation to the skin during the heating cycle can be ignored due to the large temperature gradient in the material. The cooling cycle is slow, however, and the temperature may be considered equalized through the material. Thus, radiation from both sides is allowed. Hence, the clothing heating equation under a square pulse is the same as Eq. 3.1 with $\epsilon_1 = 1 - \epsilon_2$, i.e.,

3.4 $$\rho c \frac{dT}{dt} = (1-\epsilon_2)\frac{Q_e}{t_o} - (1-\epsilon_2)\sigma(T^4 - T_o^4) \qquad 0 \le t \le t_o$$

The reader will note that the T_o^4 term is included not as a correction to the radiant term but to fix the zero point for the clothing heat capacity term on the left.

During the cooling cycle, the equation becomes

3.5 $$\rho c \frac{dT}{dt} = -2(1-\epsilon_2)\sigma(T^4 - T_o^4)$$

with $T = T_m$ at $t = t_o$, where T_m is the maximum clothing temperature, reached at the end of the thermal pulse. For the purposes of this calculation, we can set $T_o = 0$ and solve the equation with a new independent variable $t' = t - t_o$ such that $t' = 0$ and $T = T_m$ at the end of the thermal pulse.

Thus,

$$\rho c \frac{dT}{dt} = -2(1-\epsilon_2)\sigma T^4$$

or

$$+3T^{-3} = \frac{+2(1-\epsilon_2)}{\rho c}\sigma t + 3T_m^{-3}$$

or

3.6 $$T^3 = \frac{T_m^3}{1 + 2/3\,\frac{(1-\epsilon_2)}{\rho c}\sigma t T_m^3}$$

Now, the thermal input to the skin is

$$\frac{dQ}{dt} = \frac{(1-\epsilon_2)\epsilon_s \sigma T^4}{2}$$

where ϵ_s = emissivity of the skin. Thus,

3.7 $$\frac{dQ}{dt} = \frac{(1-\epsilon_2)\epsilon_s}{2}\sigma \frac{T_m^4}{\left[1 + \frac{2}{3}\,\frac{(1-\epsilon_2)}{\rho c}\sigma T_m^3 t\right]^{4/3}}$$

Since the term $2/3\ (1-\epsilon_2)\rho c \sigma$ will be of the order of 0.1 to 0.2 sec^{-1}, the cooling cycle is relatively slow.

To show how a typical burn-under-clothing calculation goes, let us consider a small bomb with Q_c = 20 cal/cm^2. We shall take the same khaki material as we used in the previous example. Thus,

$$\rho = 12\ oz/yds = 40 \times 10^{-3}\ gr/cm^2$$

$$\rho c = 16 \times 10^{-3}$$

and $$1 - \epsilon_2 = .65$$

Since the fabric heats quickly, the radiation correction is negligible and

$$\rho c \Delta T = (1 - \epsilon_2)\, Q_c = 13\ cal.$$

Thus, $\Delta T = 13/16 \times 10^{-3} = 812^\circ C$, and since the basic ambient temperature is 300°K

$$T_m = 1112^\circ K.$$

Also, the total heat input into the skin is 1/2 the stored energy in the fabric or 6.5 cal.

Now, from Eq. 3.7 one has

$$Q = 3/4\, \rho c\, \epsilon_s T_m \left(1 + 2/3\, \frac{(1-\epsilon_2)}{\rho c}\sigma T_m^3 t\right)^{1/3} = 6.5$$

whence

$$t = \frac{\left(\frac{4(6.5)}{3\rho c\, \epsilon_s T_m}\right)^3 - 1}{2/3\, \frac{(1-\epsilon_2)}{\rho c}\sigma T_m^3}$$

or

$$t = \frac{48\,(4/9)^3}{1.3 \times 1.37 \times (1.112)^3}$$

to

$$t = 1.72 \text{ seconds,}$$

Thus, the depth of burn will be the same as that from a weapon of about 40 KT on the bare skin. Referring to our previous calculations, the depth of burn is

$$\delta = 1.15 \text{ mm}$$

For 1 KT and 20 cal/cm^2, the depth of burn would have been, for bare skin,

$$\delta = 1.45 \text{ mm}$$

Thus, the clothing offers little protection and this is in accordance with experimental observations (see Ref. 6).

At lower values of Q_c, the burn depth with and without clothing is very nearly the same. Thus, summer clothing offers little better protection than bare skin.

It is clear from the above discussion that a knowledge of ignition temperatures is essential to a complete treatment of the clothing problem. Further, the ignition temperature of a given material will depend on moisture content (or relative humidity if the material has been in a fixed environment). Thus, a complete discussion of the clothing problem cannot be made until ignition temperatures are available.

Section 4. Clinical Aspects of Burn Injury; Incapacitation Times; Repair Times; Prognosis

It is generally conceded that burn injuries will play a major role in atomic warfare. The clinical aspects of burn injury are, however, easy for the layman to understand and consequently the burn problem can easily be outlined for operational personnel of the services. Moreover, the understanding of this problem is useful and important to operational commands.

Burns are usually classified as 1st degree, 2nd degree and 3rd degree. These classifications are, however, related to actual depth of injury in a general way only. Thus, the first degree burn is entirely superficial with injury confined to the epidermis which is about 0.1 mm thick. Clinically an ordinary sunburn would be classified as first degree.

The second degree burn is one in which tissue necrosis extends through the epidermis into the dermis. Since the dermis is, on the average, about 2 mm thick as against an epidermal thickness of 0.1 mm, the classification of 2° is a loose one. For example, in Section 2, we have defined a 2° burn statistically as one which extends through half the dermis. Clinically such a burn usually appears as a mottled red moist surface with or without blisters. The 2° burn area is painful and sensitive to air. This clinical appraisal of second degree burns, however, is not a good measure of burn depth. It has been useful in medical diagnosis because most of the patients seen clinically receive 2° burns in the same way. Thus, the 2° accidental burn is usually caused by short exposure to flash heat or to hot liquids and consequently there is some correlation between surface appearance of the burn and actual depth.

The third degree burns seen clinically, on the other hand, are generally caused by direct exposure to flame or to very hot objects. The skin is dry and pearly white in appearance, or charred, and the burn area is not very painful or sensitive. The depth of burn extends through the dermis into the fatty layer below, or even through the fat to the muscle. Since the entire skin tissue is lost, grafting is required for proper healing of a 3° burn. Electrical burns are almost invariably 3rd degree, but these burns should be considered separately since the heating phenomenon is quite different from any other type of burn.

The important thing to notice here is that clinical burn observations deal with the usual everyday accident. Flash burns of the duration associated with atomic weapons are seen rarely if ever and consequently the estimates of burn severity by surface appearance will not hold, in general, for atomic flash burns. Third degree burns from flaming clothing ignited by the thermal field of an atomic weapon and other indirect flame burns will probably correlate with clinical experience.

Finally, as we have seen in Section 1, (see Table 8) direct thermal burns from atomic weapons will be, in general, deep dermal burns. Third degree burns of this type will be rare and probably will be associated with fatality either from the burn or from associated blast and nuclear injury.

1. Incapacitation from Burns.

Incapacitation from thermal injury occurs from three general causes or a combination of these causes:

1. Severe burns to critical areas such as the face, hands and legs.
2. General area burns which result in early hypovolemic shock.
3. Late incapacitation from local or systemic infection.

In discussing the time to complete incapacitation, we are limited to clinical experience, plus mathematical estimates of the time to hypovolemic shock versus depth and area of burn as given in Section 2. We shall use the clinical estimates of Colonel Vogel, Chief Surgical Research Team, Brooke Army Medical Center plus some general considerations given in Ref. 9.

First, the time to hypovolemic shock as calculated in Section 2 may be considered as reasonably realistic. For example, referring to page 20 of Ref. 9, an intravenous cannula is used for fluid administration to all persons with 20% or more general body area burn. According to Table 1, Section 2, this corresponds to shock in 17 hours for the median 2° burn and shock in 12 hours for the very deep dermal burn. Thus, the calculations predict that fluid administration at this burn level is essential and this prediction correlates with clinical experience. Again, for example, a patient can survive without shock for 17 hours with a 10% full thickness burn according to the calculations of Section 2. This means that oral fluids would bridge the gap without difficulty unless nausea is a problem. Thus, the calculations of Section 2 are reasonable as compared to clinical experience. Moreover, the calculation shows the extreme seriousness of the 50% median 2° burn which would result in shock in 6 hours without fluid therapy.

Under disaster conditions, intravenous therapy would not be practical for many patients. Thus, the course of the burn trauma in untreated personnel will be a serious problem facing both medical and operational staffs of military units and installations. Proper therapy for the critically burned person calls for intravenous administration of 1.5 cc's electrolytic solution and 0.50 cc colloid solution per kg body weight per % body area burned during the first 24 hours. During this same time period, 2000 cc isotonic dextran solution is given to cover insensible losses (perspiration, breath, urine, feces). One half of the above figure is given during the second twenty-four hours.

This rule of therapy holds up to 50% area burns, above which additional fluids are generally not recommended although the physician may, by choice, choose to give more.

Thus, under disaster conditions, most patients in need of intravenous therapy will remain untreated except for administration of oral fluids. An oral solution of 3 to 4 grams salt (1/2 teaspoonful) plus 1.5 to 2.0 grams sodium bicarbonate (1/2 teaspoonful) per quart of water is surprisingly effective if the patient can tolerate the solution without vomiting. Nausea and vomiting will probably be the rule rather than the exception under conditions of atomic disaster, however. Thus, the medical and command staff will have a large number of essentially untreated persons on hand who may be expected to go into shock. The cerebral anoxia prior to shock results in restlessness often followed by mania. Also, the untreated person becomes very thirsty and, if water is consumed in large amounts, water intoxication (excessive alteration of the normal blood plasma ion concentration) occurs resulting in much the same symptomology. The burn disaster problem is thus a problem for both medical and operational commands. It could have a disastrous effect on general morale, and consequently one must decide in advance which untreated patients should be kept under heavy narcotic sedation, which should be self-treated with oral fluids, and so forth. The short discussion above is given to indicate the importance of indoctrination of command personnel as to the problems to be expected in thermal disaster. The table of Section 2 gives a rough estimate of when various patients will be in a critical medical condition and this table combined with the depth of burn estimates of Section [illegible] should provide a basis for the evaluation of the seriousness of a given disaster.

Now, for clinical estimates of time to incapacitation, we shall first consider burns to the face, backs of hands and legs. The data given may be taken as reliable.

1. 2^0 burns to the face: edema is early and rapid (1 to 3 hours). The eyes swell shut and the mouth cannot be opened (6 hours or less). A tracheotomy is generally necessary well prior to the six hour period. Thus, the facial 2^0 burn is quite serious, requires expert surgical attention and results in incapacitation in 3 hours or less.

2. Backs of hands: the skin on the backs of the hands is relatively thin. Unfortunately, there appear to be no numerical data on the skin thickness in this area or in other critical areas. However, a full thickness burn generally results in destruction of the neuro-vascular bed and causes immediate incapacitation. Probably if one estimates the burn depth required for this sort of burn, one millimeter would not be unreasonable. 2^0 burns to the backs of the hands result in incapacitation in about one hour.

3. Legs: 2^0 burns to either the front or back of the legs result in loss of use in 2 to 3 hours. Since it is unlikely that the legs alone will be burned, additional area burns will result in early onset of shock as given in Section 2.

For general area burns, it is estimated that a 15% body area burn can be tolerated indefinitely so long as the above critical areas are not extensively involved and so long as oral isotonic fluid is available in a reasonable length of time after exposure. The psychologic relation of nausea to atomic disaster is probably a major but unpredictable factor.

2. Recovery Time.

Any serious burn of the type described above, in which the individual suffers incapacitation in the first 24 hours or needs rather extensive oral fluid therapy in order to operate in the first 24 hours, is a slow healing affair by time standards of atomic warfare. Thus, the patient requires prophylactic administration of penicillin in the first five days to prevent invasive infection (septicemia) from penicillin sensitive strains of bacteria such as the β-hemolytic streptococcus strain A. During the time period 3 to 6 days, he is in maximum danger of local or invasive infection from penicillin resistant strains of staphylococcus and from such gram negative bacteria as the pseudonomus. Even without infection, the healing time of a serious burn (medium or deep dermal) must be reckoned in weeks. Thus, from a practical standpoint, the burn incapacitated person is "out" of the war and the less seriously burned patient may succumb to infection. The person with a minimal burn, say 15% area not involving critical regions, may continue to operate indefinitely if infection is prevented.

APPENDIX A

PHYSICAL AND PHYSIOLOGICAL FACTORS IN
SKIN RESPONSE TO HEATING

PHYSICAL AND PHYSIOLOGICAL
FACTORS IN SKIN RESPONSE TO HEATING

The purpose of this Appendix is to give a brief discussion of some factors in skin heating which have not been explored adequately experimentally. Henriques (Ref. 1) has done a series of experiments on the creation of thermal burns by exposure of the external surface of the skin to a constant temperature water heat source. This series of experiments has been described earlier in the text and it has been noted that Henriques' general conclusions do not hold for short periods of thermal heating, i.e., for sufficiently high rates of heat input. One wishes to know in detail why this is so and also whether there is an artifact in the Henriques' data due to skin cooling at low heat input rates.

First of all, it is well known that the skin does not act passively towards a change in external temperature. Thus, a sudden increase in skin temperature induces vasodialation and consequent increased cooling followed by a slower cooling reaction in the form of sweating. It is clear, of course, that the ability of the skin to cool has some sort of a maximum capability limited by the actual total physical capability of the skin cooling mechanisms. The total cooling capability may be further limited by the way in which the body control mechanism desires to act, or is designed to act.

Thus, for example, one may take water at 52°C (our critical temperature) and apply it to the leg above the knee via a wet cloth saturated with the hot water. A fast reading thermometer under the cloth records a temperature of only 38°C, showing that the skin cooling reaction is rapid and affective even at this high applied surface temperature. This particular experiment is extremely crude and gives little more indication than that the skin reacts quickly and cools adequately for a few seconds. We need general and accurate experimental data in order to find whether the skin chooses to or can continue this cooling rate. It seems clear, however, that the skin reaction is dominated by two general conditions:

1. The maximum physical cooling rate, and
2. The general elevation in blood temperature which results from heating of large areas of the skin.

The latter consideration must certainly be present in the body control mechanism since a general elevation of blood temperature by about 5°C can cause brain injury. Thus, one would expect the body to sacrifice the skin area in preference to accepting a high level of blood temperature. This, however, is speculation.

In any event, there must be some thermal lag time in the Henriques' data and this lag time may play a role in altering the apparent excitation energy of 150,000 cal/mole which Henriques measured. We cannot say, at the present, whether the lag time will invalidate Henriques' general conclusion. It is possible, for example, that the cell death noted by Henriques, for very long heating times, was not caused by increased temperature at all but by some other mechanism influenced by the cooling process. It is again possible that, after sufficient exposure to low heat levels, the body control mechanism begins to treat the situation as "normal" and "turns off" the attempt to cool. One can only speculate; but it is again clear that data are needed to clarify the situation.

It might be remarked that application of 52°C water to the skin is painful and one cannot go much higher without anesthesia. Thus, experiments on anesthetized animals are indicated to establish the rise in skin temperature versus time for the low heat input rates. The heat input rates which we are talking about here are of the order of 0.1 cal/cm^2 sec and less and hence have little relation to the high heat input rates from bomb pulses.

In conclusion, it seems clear that the critical temperature criterion which we have used applies to high heat input rates where skin cooling effects are negligible. It may also apply at the low input rates if skin cooling is corrected for; or, it may be that Henriques' theory will still hold at these low rates with a suitable correction for skin cooling; or, it may be that cell death results from other mechanisms at the low rates. In any event, there is a gap in knowledge here that should be closed.

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III. BLAST INJURY

When personnel are subjected to the blast wave from the detonation of a nuclear weapon, they can be injured directly by the associated change in hydrostatic pressure or indirectly by being thrown against stationary objects by the wind behind the shock front or by being hit by debris set in motion by this wind. In this Chapter we shall discuss the overall conditions necessary to produce incapacitating or more serious injuries to personnel exposed to the blast wave from a nuclear explosion.

It is our feeling that the dominant type of blast injury is that which results from the motion of personnel brought about by the applied wind load. Thus, Section 1. of this Chapter is devoted to a discussion of this problem. In Section 2., the direct blast injury problem is discussed.

Section 1. Translation Injury.

We shall discuss first the impact conditions necessary for serious to fatal injury. Unfortunately, this part of the problem does not yield to analytic techniques so that we must define critical impact conditions from a limited amount of experimental data. DeHaven(1) has carried out a series of experiments wherein cadavers were dropped head first on metal plates. These experiments showed that skull fracture occurred when the impact speed exceeded 13.5 ft/sec. Unfortunately, all the data is for impact speeds in excess of 13.5 ft/sec so that we cannot say that skull fracture does not occur at impact speeds less than 13.5 ft/sec. For the spinal column, White(1) deduces from the data by Ruff that an impact speed of 8 ft/sec is sufficient to produce fracture when impact occurs in the sitting position. On the basis of DeHaven's and Russ' data, White suggests that 10 ft/sec is the critical impact speed for serious injury to personnel for impacts against hard surfaces. It seems self-evident that orientation at impact will play an important role in the criteria for injury resulting from impact with rigid objects; however, data on this subject are not available. Accordingly, we shall use White's critical impact speed of 10 ft/sec as the impact speed against very hard surfaces in any orientation which is necessary to produce serious to fatal injury. It is expected that higher impact speeds will be necessary to produce serious injury against softer surfaces, such as ground, walls, etc. On the average, an impact speed of 20 ft/sec would seem more reasonable.

We shall now consider the qualitative features of translation motion in terms of environmental conditions. For personnel in the open (except in city streets), it appears that impact with rigid objects other than the ground is very unlikely since the total displacement, even for very high speed winds, is relatively small unless there is a lifting force from the blast wave. From some work by Taborelli, et al[2] with dummies in the 1957 Nevada Test Series, the total displacement for a standing anthropometric dummy was approximately 260 feet when the peak dynamic pressure was 15.4 psi. The dummy in this case was not only exposed to an exceptionally high dynamic pressure but he was also suspended from the head and this mode of suspension gave the dummy an initial lifting force. At lower dynamic pressures, i.e., 0.7 psi, even with head suspension, the displacement was only 20 feet. Later on we shall show that if no lifting forces are present, the man will hit the ground after his center of mass has traveled about 5 feet. Thus, the problem of collision with obstacles cannot be a great one for personnel in the open.

It appears therefore that injury to personnel in the open will occur only if the vertical impact speed is in excess of the critical impact speed discussed earlier.

For personnel in structures of various sorts, the situation is quite different. Here impact with rigid objects such as walls, doors, columns, etc. is important. In this case, however, we shall be concerned with motion over short distances.

The motion of personnel exposed to the blast wave may be divided into three parts: a horizontal motion, which depends on drag, a vertical motion which depends on gravity and to a small extent, perhaps, on lift forces, and a rotational motion. Since rotational speed can contribute to the impact speed of parts of the body, we shall be concerned with the vertical and rotational motion of personnel in the open and the horizontal and rotational motion for personnel in structures.

Thus, we shall be interested in how far the man is translated before he hits the ground, the horizontal speed he attains at any given distance up until the time he hits the ground and how hard he strikes the ground or floor. For a clean blast wave, one may make a reasonable analysis putting in the normal and frictional forces on the feet. For precursor waves, there is considerable evidence that the center of pressure is initially rather low and that the man will be lifted initially and thus will be subject only to aerodynamic forces. This lifting effect will probably be present only for personnel in the open and not for personnel in buildings. Thus, we wish to consider two general cases; in the first case the forces on the feet of the standing man will play a major role, in the second case the man will be subject to aerodynamic forces only.

1. Translational and Rotational Injury to Standing Personnel for a Clean Blast Wave.

It was mentioned in the introduction that aerodynamic torque plays no appreciable role in the motion of personnel subjected to blast waves. We wish to consider the experimental evidence for this conclusion and show that rotation is induced by initial frictional forces only. We shall then calculate the motion of a standing man and consider rotation induced by frictional forces.

Displacement experiments with anthropometric dummies have been carried out in a bomb field test. Only one experiment contains a sufficient amount of information for analysis. In this experiment, both a prone and standing 165 pound dummy were subjected to the wind loading from a nuclear explosion wherein the peak dynamic pressure was .7 psi. The prone dummy was not moved while the standing dummy was displaced 21.9 feet and attained a maximum speed of 21.4 ft/sec. It should be noted that the dummy contacted the ground in a little more than 13 feet and slid the remaining distance. During this motion, the dummy rotated feet first through an angle of approximately 120° in such a way that its head hit the ground first. The experimental situation for the standing dummy differed significantly from that for a standing man in the same situation in that the dummy was supported by the head with a negligible load on its feet, whereas for a standing man, the entire load is supported on his feet. We will show that the rotation of the dummy involves no aerodynamic torque and that the motion in this case is explained by the frictional retarding force on the dummy's head during the initial stages of motion. For the standing man, the frictional retarding force will be on his feet so that he will rotate head first.

In the experiment mentioned above, a 160 pound anthropometric dummy was suspended by the head with only a slight load (less than 6 pounds) on his feet and then subjected to the wind from a nuclear explosion. Figure 1 shows a plot of the rotation of the dummy versus time, the point P on the graph corresponding approximately to the time at which the dummy's head was released. From about $t = .15$ sec on θ varies almost linearly with time so that

$$\frac{d\theta}{dt} = \text{constant}$$

and

$$\frac{d^2\theta}{dt^2} = 0$$

Thus, for $t \geq .15$ sec, the dummy is subjected to no torque. The initial motion of the dummy can be accounted for by torque due to the head support. If F is the frictional retarding force on the head, then neglecting aerodynamic torque, we have

$$\left.\begin{aligned} I\frac{d^2\theta}{dt^2} &= F\ell \sin\theta \\ &= 0 \end{aligned}\right\} \quad \text{when} \quad \left\{\begin{aligned} &0 \leq \theta \leq \theta_c \\ &\theta \geq \theta_c \end{aligned}\right\}$$

where ℓ is the distance from the center of gravity of the body to the point of attachment on the head support, and θ is the angle between a line through the head and feet of the body when it is erect and vertical to the ground. Now, let

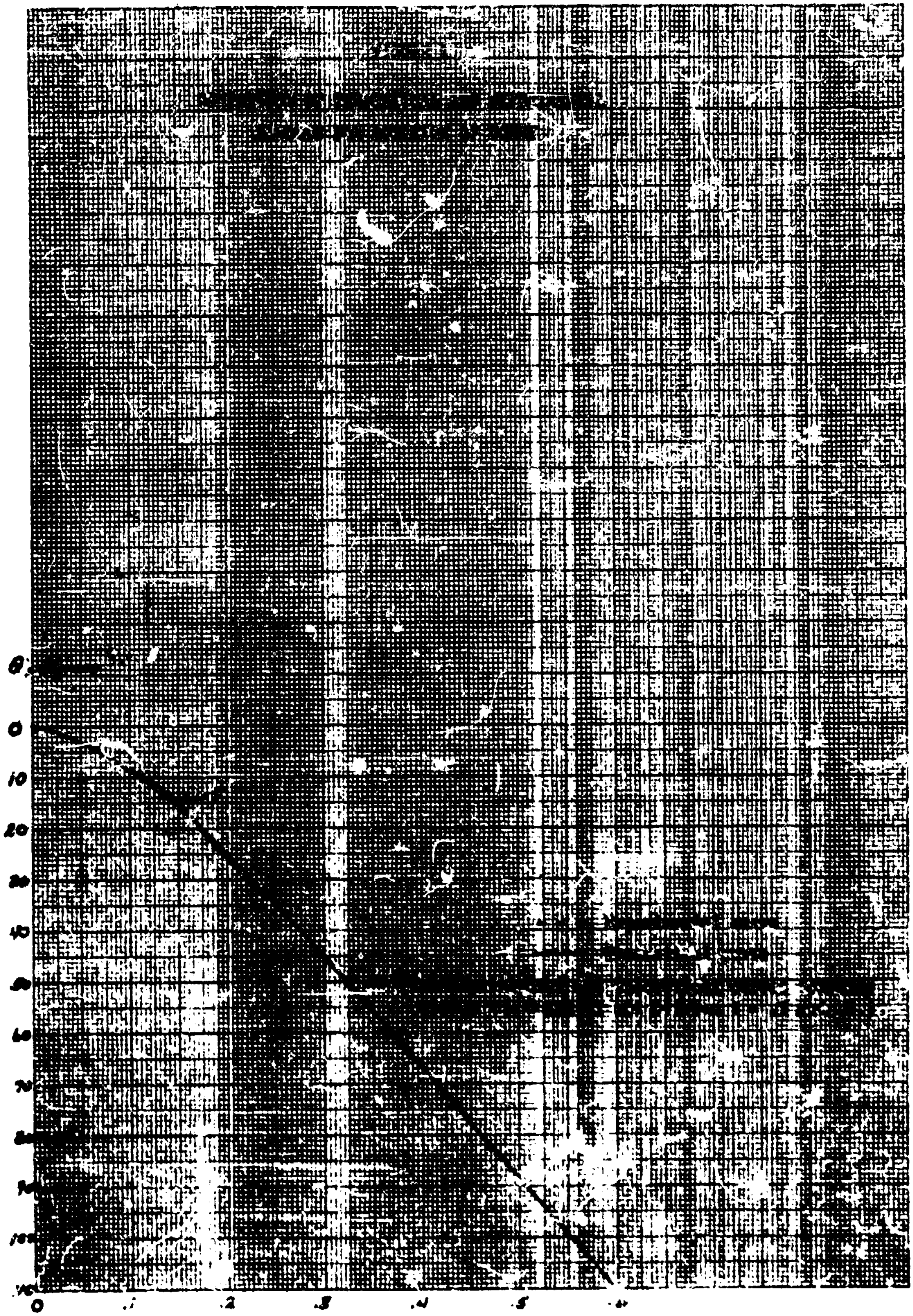
$$\omega = \frac{d\theta}{dt}$$

so that

$$\omega\frac{d\omega}{d\theta} = \frac{d^2\theta}{dt^2}$$

We then have

$$I\omega\frac{d\omega}{d\theta} = +F\ell\cos\theta$$


0
10
20
0
.1
.2
.3
.4
.5
.6
Time seconds

Thus,

$$\frac{1}{2}\omega^2\Big|_{t=0}^{t=t_0} = +\frac{F\ell}{I}\sin\theta + C_0$$

when $t = 0$, $\theta = 0$ and $\omega = 0$, so that

$$\frac{1}{2}\omega^2(t) \quad \frac{F\ell}{I}\left[1+\sin\theta\right]$$

Thus,

$$\omega t = \frac{d\theta}{dt} = \left[\frac{2F\ell}{I}\right]^{1/2}\left[1+\sin\theta\right]^{1/2}$$

and integrating, one finds

$$\frac{\sqrt{2}+\sqrt{1-\sin\theta}}{\sqrt{1+\sin\theta}} = e^{C_0}e^{-\sqrt{2}\sqrt{\frac{F\ell}{I}}\,t}$$

Now, when $t = 0$, $\theta = 0$, so that $e^{C_0} = \sqrt{2}+1$. Thus, for $t \leq .15$ sec, θ depends on the time according to

$$\frac{\sqrt{2}+\sqrt{1-\sin\theta}}{\sqrt{1+\sin\theta}} = \left[\sqrt{2}+1\right]e^{-\sqrt{2}\sqrt{\frac{F\ell}{I}}\,t}$$

If we assume that the man has the same moment of inertia as a right circular cylinder of radius .75 ft and length 6 ft, then we can satisfy this equation at $t = .15$ sec and $\theta = 15^\circ$ by taking $f = .9$ where f is the coefficient of moving friction between the dummy's head attachment and the folding frame. The resulting curve is shown plotted in Figure 1 along with the experimental curve. The agreement is good. The point here is that the data are satisfied with reasonable assumptions concerning the moment of inertia of the man and the initial frictional retarding forces. Thus, as we said earlier, the rotational motion of the dummy can be explained on the basis of no aerodynamic torque. We can therefore conclude that the rotational speed of a standing man who is subjected to a high speed wind will be determined by the initial frictional retarding forces on his feet and that this speed will be independant of the wind speed.

We turn now to the theoretical calculation of translation and rotation of a man under applied wind forces and retarding frictional forces.

Let us consider first the speed with which the man's head strikes the floor or the ground. Since the center of pressure of the man will coincide roughly with his center of mass, the rotational torque about the center of mass is supplied by the ground forces alone. Thus, let

m = mass of the man

ℓ = height of the man

I = moment of inertia of the human body about the center of mass

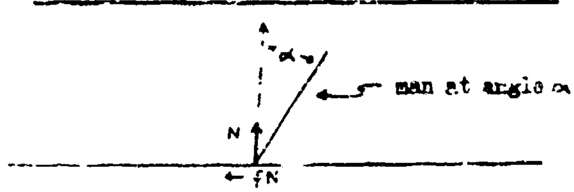
α = angle between the body axis and the vertical

N - normal force on the feet (exerted by the ground on the feet)

fN = frictional force exerted by the ground on the feet, where f is the coefficient of friction.

First we note that if the initial dynamic pressure is above 0.2 psi (suddenly applied) and if $f \simeq 0.8$, the man will slide on his feet as he rotates. Thus, in any important case, we may consider the man to be sliding. Referring to the figure below, however, we have for

Man Rotating and Sliding Under Wind Forces



rotation about the center of mass

(1) $$I\ddot{\alpha} = \frac{N\ell}{2}\sin\alpha + \frac{fN\ell}{2}\cos\alpha$$

and for vertical motion of the center of mass

(2) $$m\ddot{y} = mg + N$$

The right hand side of (1), however, is $N\ell/2\left[\sin\alpha + f\cos\alpha\right]$ and the factor in brackets varies from a value of f at $\alpha = 0$ to $1+f/\sqrt{2}$ at $\alpha = \pi/4$ to 1 at $\alpha = \pi/2$. Thus, one may, for sufficient accuracy, replace the right hand side by the average value of $\sin\alpha + f\cos\alpha$. This average value is

$$\bar{\lambda} = \frac{2}{\pi}\int_0^{\pi/2}(\sin\alpha + f\cos\alpha)\,d\alpha = \frac{2}{\pi}\left[1+f\right]$$

or for $f = 0.8$

$$\bar{\lambda} = 1.14$$

Thus, one has, to good approximation,

$$I\ddot{\alpha} = 1.14\,\frac{N\ell}{2}$$

and $m\ddot{y} = -mg + N$ or $N = m\ddot{y} + mg$. Substituting the value of N from the second equation in the first, one has successively

$$I\ddot{\alpha} = 1.14\,m\frac{\ell}{2}\left[\ddot{y} + g\right]$$

(3) $$I\dot{\alpha} = 1.14\,\frac{m\ell}{2}\left[\dot{y} + gt\right]$$

and

(4) $$I\alpha = 1.14\,\frac{m\ell}{2}\left[y + \tfrac{1}{2}gt^2\right] - 1.14\,\frac{m\ell^2}{4}$$

since at $t = 0$, $\dot{\alpha} = \dot{y} = 0$ and $y = \ell/2$.

Thus, setting $I = m\ell^2/12$, (3) and (4) become, respectively

(5) $$\dot{\alpha} = \frac{6.84}{\ell}\left[\dot{y} + gt\right]$$

and

(6) $$\alpha = \frac{6.84}{\ell}\left[y + \tfrac{1}{2}gt^2\right] - 3.42$$

Now, the man's head strikes when $\alpha = \pi/2$ and $y = 0$. This yields $t = 0.05$

seconds for the average man (i.e., $\ell \approx 67"$).

Since, however, $y = \ell/2 \cos\alpha$, $\dot{y} = -\ell/2 \sin\alpha \;\dot{\alpha} = -\ell/2\dot{\alpha}$ at $\alpha = \pi/2$. The speed with which the head hits is, therefore,

$$v_H = \frac{\ell}{2}\dot{\alpha} + |\dot{y}| = \ell\dot{\alpha}$$

Thus, from (5), $\ell\dot{\alpha} = 24.5$ ft/sec $= v_H$.

In other words, the man strikes with sufficient speed to cause serious injury or death.

2. Translation Under Wind Forces and Frictional Forces; Derivation of the Corrections to the Head Speed for Rotation and Frictional Effects.

Having considered the problem of the speed with which the man hits the ground, we must next consider his net translational speed at time prior to hitting the ground or floor. The translational speed of his head will be made up of the speed acquired by his center of mass under wind forces plus the rotational component tangent to the ground. For complete discussion of this problem, we need α, $\dot{\alpha}$, and N as functions of time t. These quantities, as calculated from the foregoing equations are shown in Table 1. The horizontal head speed $v_{RH} = \ell/2\,\dot{\alpha}\cos\alpha$, due to rotation alone, is also needed. This is also shown in Table 1. We note that the head speed $\ell/2\,\dot{\alpha}\cos\alpha$, due to rotation alone, reaches values as high as 8 ft/sec at early times.

Table 1

Values of the Translation Parameters, α, $\dot{\alpha}$, N/mg and $\ell/2\,\dot{\alpha}\cos\alpha$

α	t (sec)	$\dot{\alpha}$ (rad/sec)	N/mg	$\ell/2\,\dot{\alpha}\cos\alpha$ (ft/sec)
0	0	[illegible]	1.000	0
.2	.116	2.75	.219	7.45
.4	.184	3.12	.097	8.01
.6	.246	3.31	.073	7.62
.8	.305	3.49	.077	6.78
1.0	.361	3.67	.095	5.52
1.2	.414	3.90	.125	3.95
1.4	.463	4.18	[illegible]70	1.98
$\pi/2$	.502	4.40	.226	0

Another quantity of interest is the reduction in forward speed due to frictional forces. This reduction in speed is $v_s = -g\int_0^t N/mg\, dt$, with $N/mg = \frac{1 - \ell/2 \cos\alpha\, \dot{\alpha}^2/g}{1 + 3.42 \sin\alpha}$ given in the table below. Values of v_s are shown in Table 2.

Table 2

Speed Reduction Due to Friction as a Function of Time

t (sec)	v_s (ft/sec)
0	0
.1	-1.6
.2	-2.1
.3	-2.4
.4	-2.7
.5	-3.2

Now, we wish to consider the equation of motion for translation alone under the applied wind force and the retarding frictional force. This equation turns out to be

(7) $$m\frac{d^2x}{dt^2} = \tfrac{1}{2} A C_d \rho\left(t - \frac{x}{U_o}\right)\left[\frac{dx}{dt} - u\left(t - \frac{x}{U_o}\right)\right]^2 - f N(t)$$

where

m = mass of the man

A = mean presented area

C_d = drag coefficient

ρ = density of air at point (x,t)

u = wind speed behind shock at point (x,t)

x = man's displacement from his position at the time of shock arrival.

Equation (7) will be derived later (3. Pure Translational Motion Under a Blast Wave) where it will be seen that, due to motion of the shock front relative to the ground, $\rho(t - x/U_o)$ and $u(t - x/U_o)$ appear in the exact equation rather than $\rho(t)$ and u(t). It will also be shown that use of $\rho(t)$

and u(t) rather than $\rho(t - x/U_o)$ and $u(t - x/U_o)$ results in negligible error. Here our purpose is to consider the solution of (7) for very short distances of translation and also to consider the way in which velocity increase due to rotation and decrease due to friction may be corrected.

Now, as a first case, let us consider the specific problems of personnel who are subject to blast translation in rooms of buildings or in city streets. Since the individual involved either will hit the floor or pavement at critical speed in about 1/2 second or strike an obstacle prior to hitting the floor or pavement, we are interested in his motion over a very short period of time. As we shall see, the time period of interest is generally 1/4 second or less. Thus, for almost any size of bomb, and certainly any bomb size which might be used against populated areas, the time period of interest is very short compared to the blast wave duration. Hence in Eq. (7) we shall have $u \gg dx/dt$ and $u \approx u_o$, $\rho \approx \rho_M$ over the period of interest, where u_o is the peak wind speed and ρ_M is the peak density behind shock.

Under these circumstances, one has

$$(8) \qquad m\frac{d^2x}{dt^2} = \tfrac{1}{2}\rho_M A C_d u_o^2 = q_m A C_d$$

where q_m = peak dynamic pressure. For calculation purposes we take

$$mg = w = 154 \text{ pounds}$$

$$A \approx 4 \text{ ft}^2$$

and $\qquad C_d \approx 1$

Integrating (8), one has

$$(9) \qquad m\frac{dx}{dt} = q_m A C_d t$$

(10) $$m x = \rho_m A C_d \frac{t^2}{2}$$

We set dx/dt = 20 ft/sec, the critical speed, in (9) and calculate t. We then calculate x from (10). This leads to the following table, for the case of clean shocks.

Table 3

Time and Distance Required to Reach the Critical Speed of 20 ft/sec for Various Shock Overpressures

Overpressure (psi)	ρ_m (psi)	t (sec)	x (ft)
30	17	.0098	.097
20	8.2	.0202	.210
10	2.2	.0754	.681
5	.60	.278	2.77
4	.37	.449	4.48
3	.20	.834	8.34

Inspection of Table 3 shows that 5 psi overpressure or more is a good criterion for death or serious injury from translational injury for personnel in buildings. The reason for this is that, using Tables 1 and 2 and including both the head speed due to rotation and the retarding speed due to friction, a man struck by the 5 psi wave will reach the critical speed in 0.2 seconds in a distance of 3 feet. Thus, all personnel at greater distances than 3 feet from a wall either have critical speed upon impact with a wall or hit the floor at critical speed. At 4 psi, on the other hand, personnel cannot reach critical speed either by hitting the wall or the floor unless they are about 8 feet from the wall. One must remember that the head moves about 2.7 feet more than the center of gravity in the critical instance where the head hits the wall and floor at the same time. Eight feet is a rather large average distance of motion for the ordinary room. Thus, one can set 5 psi as a reasonable overpressure for serious injury or death for personnel in rooms.

For personnel in city streets, however, the hardness of the pavement and vertical obstacles and the relatively large average distances of free translation indicate selection of a lower overpressure. Since the critical overpressure to just cause sliding under frictional forces is 3.0 psi or a q_m of 0.2, it seems reasonable to set this overpressure as the critical one for personnel in city streets. It may be argued, of course, that people will be knocked to the pavement, without sliding, at lower overpressures with sufficient speed for serious injury. The question of reaction time of the individual comes into play, however, and one must judge whether he can use his hands to cushion the fall. Three psi seems the best choice.

3. Pure Translational Motion Under a Blast Wave.

Finally we wish to discuss the translational problem under the assumption that the man is lifted by the blast wave initially and there is, consequently, negligible interaction with the ground prior to the time that he hits. We have just seen, from analysis of the dummy experiment, that rotational effects do not play a significant role unless there is interaction with the ground or with a head support. The problem will then be one of pure translation except, perhaps, for a small rotational speed induced at the start of the motion.

It should be noted that the quantity of interest measured in field tests is the dynamic pressure, or $\frac{1}{2}\rho v^2$. ρ and v are not measured independently and it is difficult to do so. If the shock is clean, however, ρ and v may be inferred independently on theoretical grounds. In the case of a precursor, the inference is difficult. In any event, the theory requires an independent knowledge of ρ and v for a precise solution. In Appendix A it is shown that the wind speed behind shock varies to a very good approximation as $u = u_0 \epsilon^{-t/t_+} (1 - t/t_+)$. As for the variation of ρ behind shock, it is sufficient to use the average value $\bar{\rho}$ since we must assume average values for presented area and for drag coefficient.

We will phrase the problem exactly in the sense that the motion of the shock front relative to the ground and the motion of the translated object relative to the wind will be written down exactly, using only an assumption of constant mean presented area of the object and constant drag coefficient.

Let x be a coordinate fixed relative to the ground and let the shock travel out the positive x-axis. We shall let the shock be $x = 0$ at time $t = 0$. Let x' be a second coordinate fixed with respect to the shock front so that $x' = 0$ at the shock front and $x' < 0$ inside the shock.

Now, the value of q behind the shock is measured in the field as a function of time at a fixed ground station. From this value of q, the wind speed is inferred. Thus, the measurement gives the wind speed $u(t)$, where $u(t) = u_o$ at $t = 0$, and u_o is the maximum wind speed just behind shock. It is clear from this that the wind speed at distance x' behind shock is $u_o(|x'|/U_o)$ or $u(-x'/U_o)$ (since x', as we have defined it, is negative in the shock), where U_o is the shock speed relative to the ground.

Thus, noting that

$m \equiv$ mass of the translated object

$A \equiv$ mean area of the translated object

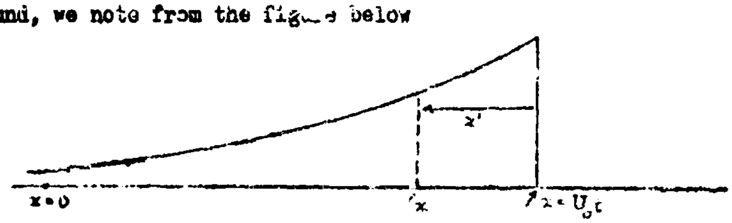
$\rho \equiv$ air density behind shock

the motion of the object in the x' system is

$$m\frac{d^2x'}{dt^2} = \tfrac{1}{2}\rho A C_d\left[\frac{dx'}{dt} + \left(U_o - u\left(\frac{-x'}{U_o}\right)\right)\right]^2 \tag{11}$$

where $u(t) - U_o$ is the motion of the air behind shock in the coordinate system fixed with respect to the shock front. The direction of $u(t) - U_0$ is along the negative x-axis, as is the direction of dx'/dt. In this coordinate system, the value of dx'/dt, as the object enters the shock front, is $-U_0$.

To write down this same equation in the system fixed relative to the ground, we note from the figure below



$$x = U_0 t + x' \qquad \text{or} \qquad x' = x - U_0 t$$

$$\frac{dx'}{dt} = U_0 + \frac{dx}{dt} \qquad\qquad \frac{d^2x'}{dt^2} = \frac{d^2x}{dt^2}$$

Thus, we find, in the coordinate system fixed with respect to the ground

$$m\frac{d^2x}{dt^2} = \frac{1}{2}\rho\left(t - \frac{x}{U_0}\right) A C_d \left[\frac{dx}{dt} - u\left(t - \frac{x}{U_0}\right)\right]^2 \tag{12}$$

In other words, the effect of motion of the shock front is to replace $u(t)$ by $u(t - x/U_0)$. If any frictional retarding forces F_s are present then F_s will be added in the right hand side of (12). The initial condition on (12) is, of course, $dx/dt = 0$ at $t = 0$.

As we mentioned before, it is sufficiently accurate to replace $\rho(t - x/U_0)$ by $\bar{\rho}$, the average value behind shock. The question arises, however, as to whether $u(t - x/U_0)$ may be replaced by $u(t)$ without significant loss of accuracy. A simple argument shows that this can be done.

One notes that, to the first order,

$$u\left(t - \frac{x}{U_0}\right) \cong u(t) - \frac{x}{U_0}\left(\frac{\partial u}{\partial x}\right)\bigg|_{x=0}$$

But, to a first approximation for clean shocks, u decreases exponentially; i.e.

$$\mu(t) \simeq \mu_o \epsilon^{-t/t_+}$$

and

$$\frac{\partial \mu}{\partial t} \simeq -\frac{\mu_o}{t_+} \epsilon^{-t/t_+} = -\frac{\mu(t)}{t_+}$$

Thus,

(13) $$\mu\left(t - \frac{x}{U_o}\right) \simeq \mu(t)\left(1 + \frac{x}{U_o t_+}\right)$$

Now, let us consider (13) for the case of translation of a man. The critical speed for injury is low so that, to the accuracy needed in estimating the correction term $x/U_0 t_+$, we may neglect the effect of dx/dt in (12). Thus, approximately

$$m\frac{d^2x}{dt^2} = \frac{1}{2}\rho A C_d \mu_o^2 \epsilon^{-2t/t_+}$$

and integrating one has, successively

$$mv = \frac{\rho A C_d \mu_o^2 t_+}{4}\left[1 - \epsilon^{-2t/t_+}\right]$$

and

$$mx = \frac{\rho A C_d \mu_o^2 t_+}{4}\left[t + \frac{t_+}{2}\epsilon^{-2t/t_+} - \frac{t_+}{2}\right]$$

But, v has reached its maximum, for all practical purposes at $t = t_+$. Therefore

$$x_{max} \simeq v_{max}\frac{t_+}{2}$$

Hence, the maximum value of the correction term in (13) is

$$\frac{x_{max}}{U_o t_+} \simeq \frac{v_{max}}{2U_o}$$

Since v_{max} is only 20 ft/sec for critical injury, $v_{max}/2U_0 <$ [illegible] even for a shock traveling at sonic speed. Thus, one may ignore the correction x/U_0 to the time t. The correction term is, in fact, [illegible] unimportant even for missiles.

Finally, therefore, in considering the free translation of men and missiles, we shall set

$$(14) \qquad m\frac{d^2x}{dt^2} = \frac{1}{2}\rho A C_d \left[\frac{dx}{dt} - u(t)\right]^2$$

where dx/dt enters now only as a correction to the wind speed behind shock and there is no time delay involved in the evaluation of $u(t)$.

Now we wish to consider the solution of (14) for the free translation of men and missiles. It is expected that $u(t)$, the applied wind load will vary with time in such a way that (14) will not be directly integrable; hence we need an approximate technique. It turns out that if $u(t)$ is linear, (14) is integrable. Thus, we shall approximate $u(t)$ by a sequence of linear strips of the form $u(t) = u_o(b - a\tau)$, where $\tau = t/t_+$, t_+ = duration of the positive phase, and u_o = peak wind speed. For each portion of the positive phase we shall use properly selected values of a, b; i.e., a_i and b_i. At the present, however, we shall carry through the integration for general values of a and b and then consider a specific case.

Now, setting $u(t) = u_o(b - a\tau)$ in (14), and letting $\varphi = u - dx/dt = u - 1/t \; dx/d\tau$, we have

$$\frac{d\varphi}{d\tau} = -a u_o - \frac{1}{t_+}\frac{d^2x}{d\tau^2}$$

Thus, (14) becomes

$$(15) \qquad \frac{d\varphi}{d\tau} + \frac{\rho A C_d t_+}{2m}\varphi^2 = -a u_o,$$

with $\varphi(0) = u_o b$. This integrates to

$$\tau = C_0 - \sqrt{\frac{2m}{\rho C_d A a u_o t_+}} \tan^{-1}\left(\sqrt{\frac{\rho A C_d t_+}{2m a u_o}}\,\varphi\right)$$

where C_0 is a constant of integration. If we set

$$D = \frac{\rho A C_d u_c t_+}{2w}$$

then

$$\tau = C_0 - \frac{1}{\sqrt{Da}} \tan^{-1}\left(\sqrt{\frac{D}{a}}\, \frac{\varphi}{u_0}\right) \tag{16}$$

Solving for φ and setting $\varphi = u - 1/t_+ \, dx/d\tau$, one has after a second integration

$$\frac{x}{u_0 t_+} = b\tau - a\frac{\tau^2}{2} - \frac{1}{D} \log\left|\cos \sqrt{Da}\,(C_0 - \tau)\right| + C_1 \tag{17}$$

where C_1 is a second constant of integration. The formulas (16) and (17) may be used for the successive integration of (14) when u(t) is approximated piece-wise by linear strips.

As an example of the integration of (14), we take the case of a clean shock where $u = u_0(1 - t/t_+)\epsilon^{-t/t_+}$. We approximate u by ten piece-wise linear strips of the form

$$u(\tau) = u_0(b_i - a_i\tau) \qquad \frac{i-1}{10} \leq \tau \leq \frac{i}{10} \qquad 1 \leq i \leq 10$$

The proper values of a_i and b_i are given in the table below.

Table 4

i	a_i	b_i
1	1.86	1.000
2	1.59	.9790
3	1.36	.9270
4	1.17	.8700
5	.990	.7980
6	.830	.7180
7	.710	.6460
8	.592	.5634
9	.491	.4824
10	.407	.4070

Equations (16) and (17) are applied piece-wise to the approximation for $u(\tau)$ to obtain $\Phi(t)$ and $x(t)$. dx/dt is then obtained from Φ and plotted against x for various values of D. The results are shown in Figures 2, 3, 4 and 5.

As we have noted before, free displacement is probably not an important consideration with personnel. Unless one ascribes an initial lifting force to the blast wave, the standing man rotates under frictional forces and hits the ground after traveling 5 feet. These free displacement curves are, however, useful for computing missile speeds and then will be useful for computation of displacement of personnel if it should happen that later analysis of weapon blast fields shows a lifting force with precurser type waves. The missile problem will be considered in more detail in the later stages of the "environmental" contract. As of now, the missile problem appears to have little importance except, perhaps, for cases of personnel confined in rooms.

To illustrate the use of the curves we shall take an example from The Dikewood Report[3]. We take the example (Page 25) of the steel spherical missile exposed to 0.6 psi dynamic overpressure. Here the drag coefficient is rather well known. It is about 0.4. The other parameters are

$$q_m = 0.6 \text{ psi}$$

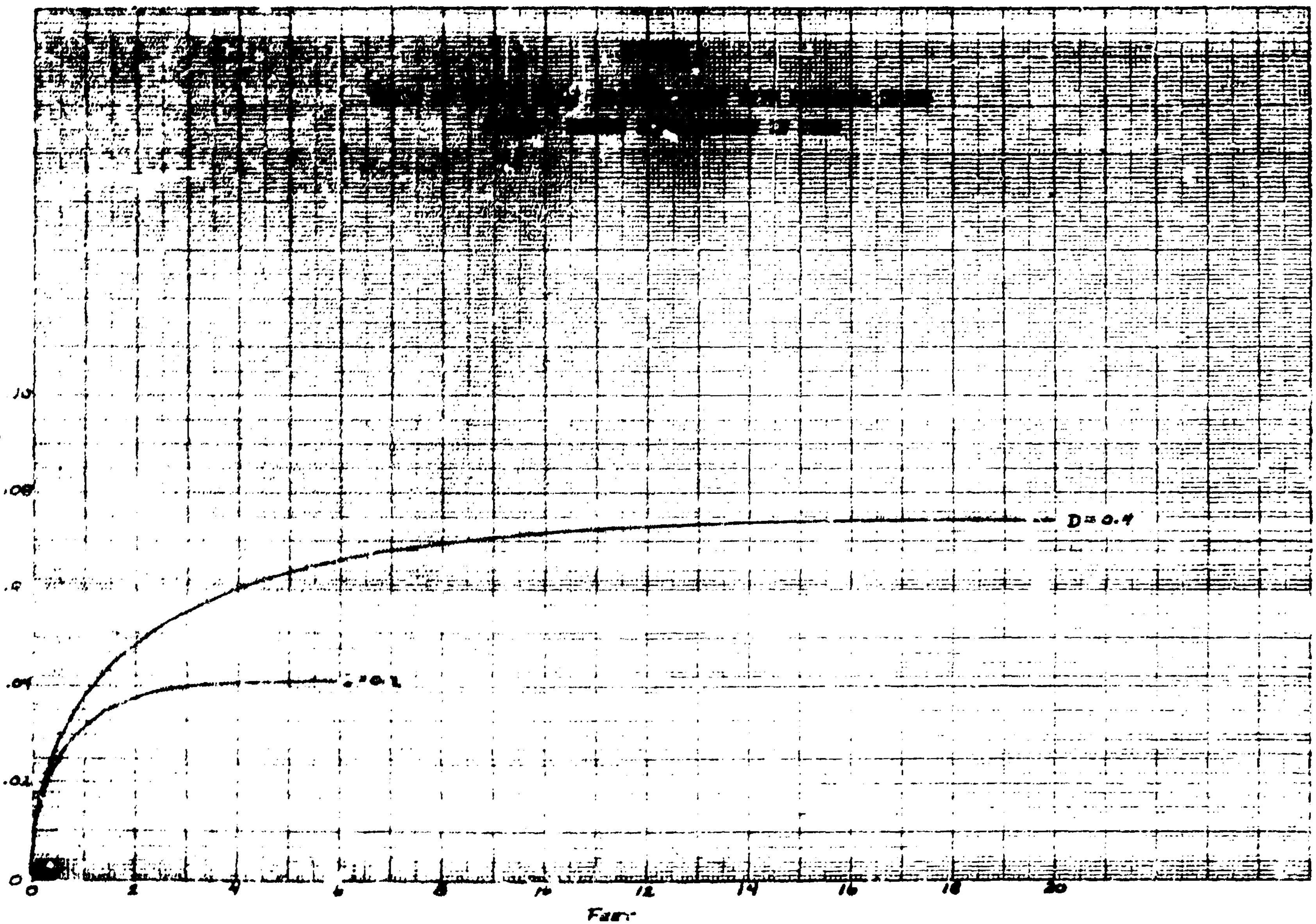
$$u_0 = 247 \text{ ft/sec}$$

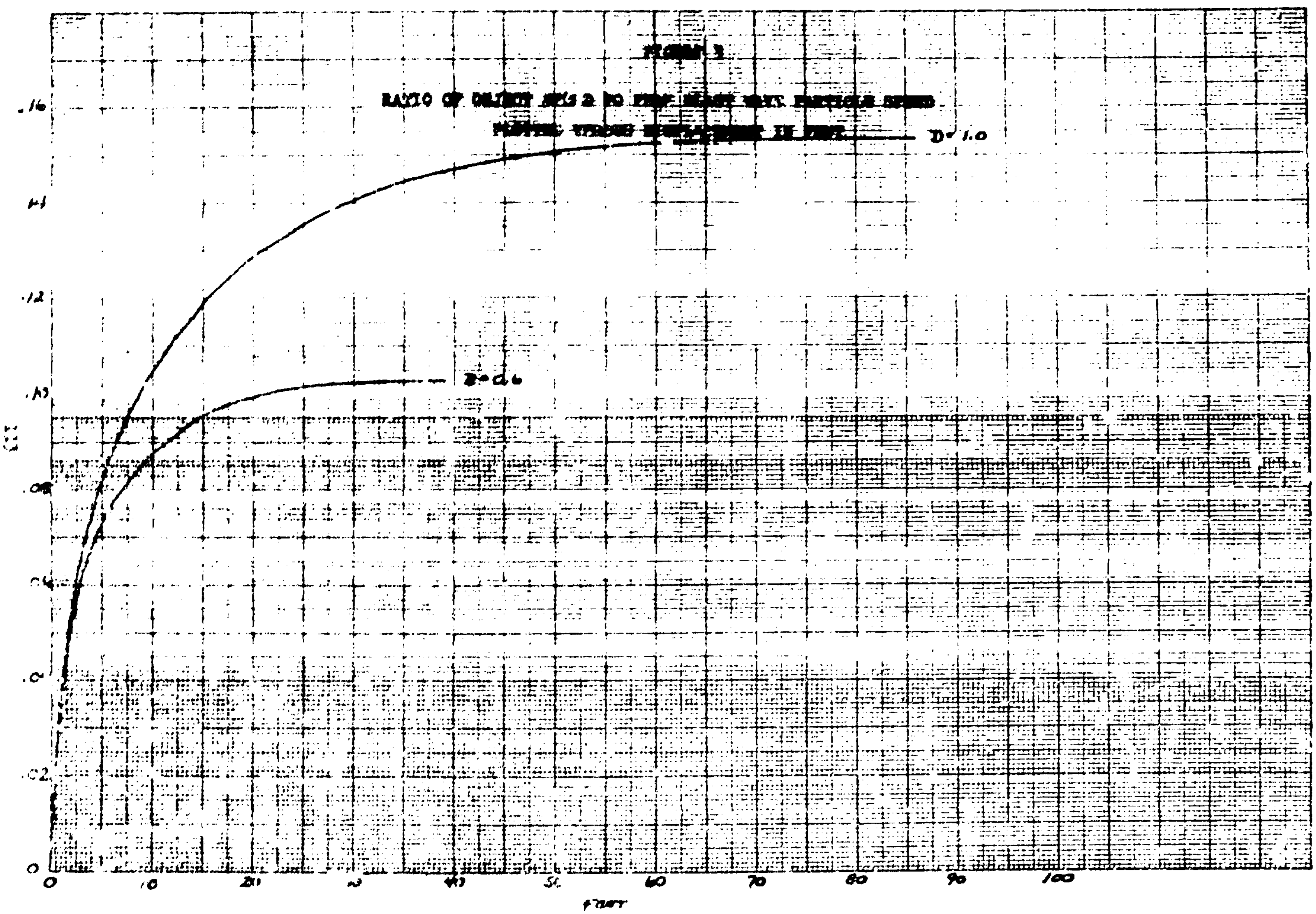
$$w = .13[illegible] \text{ grams}$$

$$\rho = .09 \#/ft^3$$

$$A = \pi r^2 \qquad r^2 = 1/16''$$

This yields D = 2.4. Referring to Figure 4, we find $(dx/dt)_{max} = .27\, u_0 =$ 67 ft/sec. The report gives an average velocity of 70 ft/sec for 5 such missiles. Since the measured speed can be as [illegible]% of the maximum speed, the greatest value of the measured speed could have been as high as 78 ft/sec. In any event, the agreement is good, perhaps fortuitously so.


.10
.08
.06
.04
.02
0
0
2
4
6
8
10
12
14
16
18
20
Feet
D = 0.4



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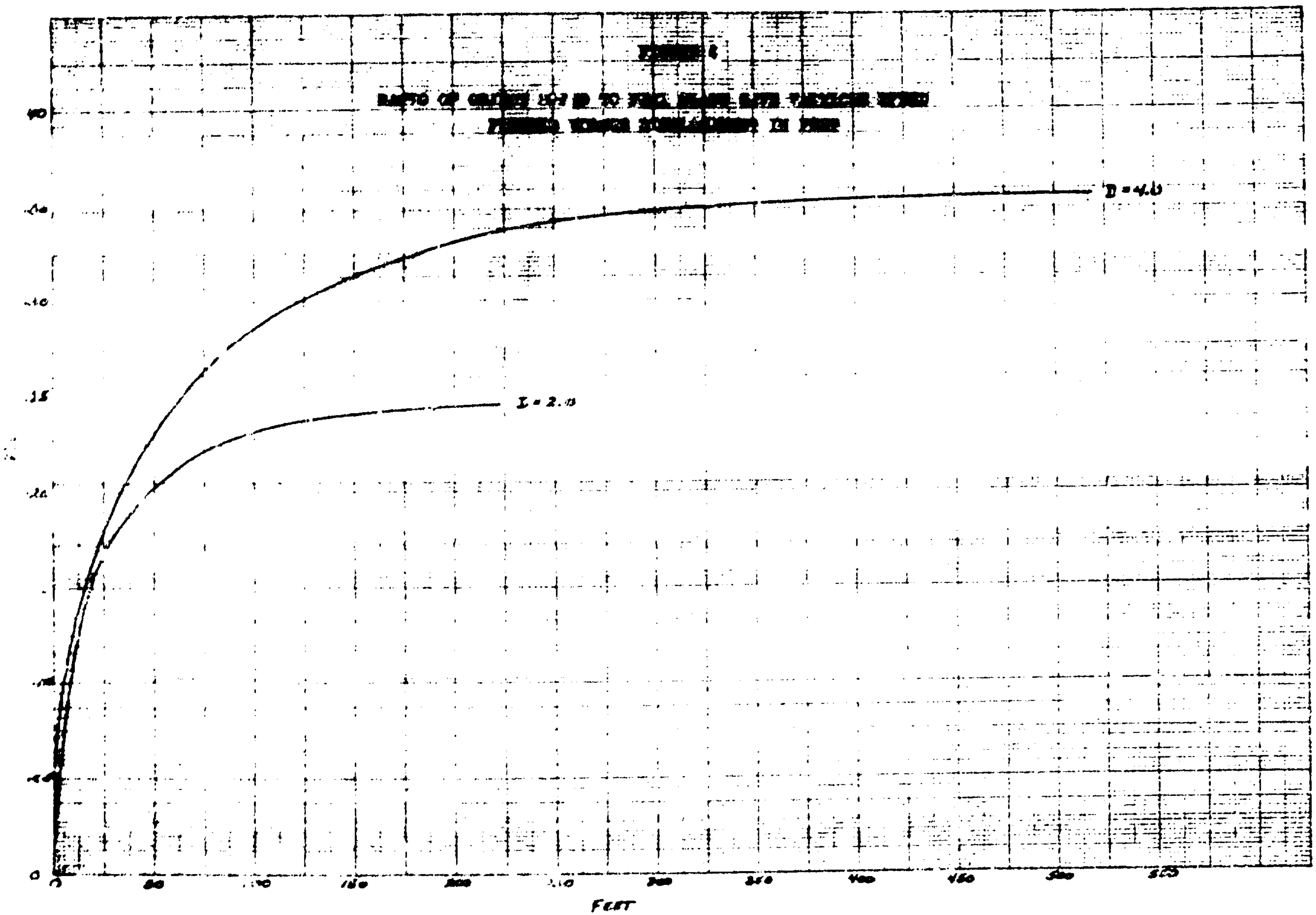

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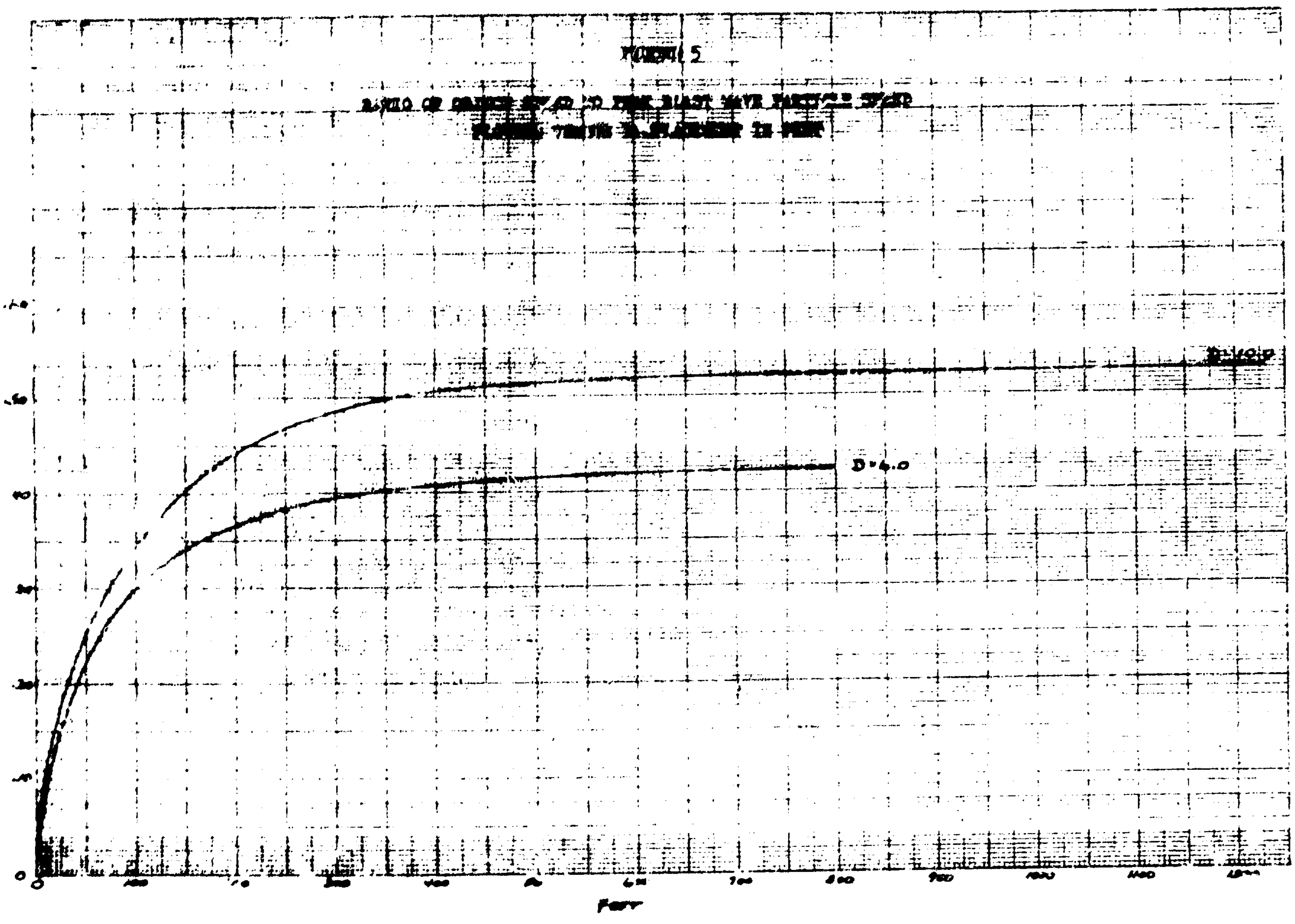

D=4.0
Feet

Section 2. Direct Blast Injury.

The bulk of the available data on direct blast injury to man and animals is laboratory data taken for the most part with mice and other small animals. The existing data for man do not contribute much to the present understanding of the mechanism of direct blast injury; however, the following two particularly important features are to be noticed. In an analysis of 200 persons subjected to the blast from a high explosive, Barrow and Rhoads(4) discovered that those persons who survived for 60 minutes recovered and that in almost every case injured persons suffered from extreme fatigue and were listless and apathetic for an extended period after the explosion. For the most part, these and other incapacitating symptoms disappeared at the end of 24 hours. Although not all exposed persons suffered serious to fatal injuries, injury to the eardrum was an almost universal effect. Deafness from such injury can, of course, persist for days to weeks but is not incapacitating for specific jobs in which hearing is not essential.

Animal data taken under controlled conditions are somewhat more illuminating as far as the physical effects of blast are concerned. For mice, rats, rabbits and dogs, the following effects of blast have been observed in almost every case:

1. Lung hemorrhage
2. Lung edema
3. Severe respiratory effects
4. Air embolism (in some cases)
5. Damage to central nervous system

6. Superficial shock

7. Increase in lung edema with oxygen treatment

8. "Rib markings"

9. A macroscopically unaffected heart

10. Dilitation in the right ventricle

11. Abdominal lesions, rupture and hemorrhage which appear to depend on the gas content of the particular organ.

12. A fairly large dependence of effect on the duration of the blast overpressure.

13. No significant effect on the chemistry of the peripheral circulatory system.

14. The failure of the severely injured animal to attempt inhalation which results in cyanosis of varying duration.

The following experiment(5) is thought to be the most indicative of the predominant effect in blast injury. In this experiment, mice are constrained to a vertically fixed flat plate and exposed to the blast from a fixed weight of high explosive at varying distances. In each case, the hemoglobin content of the lung, the lung weight and mortality versus distance are given. All mice with lung weights in excess of $\approx$ 375 mg. died and all those with lung weights less than 280 mg lived. In the region of lung weight from 280 to 375 mg, approximately 35% died. With respect to the hemoglobin content of the lungs, all those mice whose hemoglobin content was in excess of $\approx$ 8 mg. died while all those with less than $\approx$ 8 mg. lived. It should be noted that although the distance and blast overpressure were the same in many cases, death occurred only as described above. In the next experiment, the mice were exposed to blast in a shock tube where various portions of the animals were shielded. Here the lung weight after blast depends quite heavily on the region protected. When the head is shielded, the lung weight is greatly reduced from that for the unshielded animals. A thoracic shield appears slightly to increase the lung weight over that for the unshielded animals. The report(5) gives the relationship between lung weight, mortality and shielding for a large number of cases. Here the animals with head shielding survived* for the most part while all the unprotected animals and the ones with thoracic shields all died. The shields were rigid and did not contact the animal** so that the protection afforded was from the direct impact of the blast.

It appears that the above experiment combined with the lack of other general tendencies indicates that the primary effect of blast is damage to the central nervous system. Unfortunately, the animals were constrained in these experiments so that the conclusion may be restricted to this circumstance. It is mentioned in the report(4) that swollen lungs are not often observed in unconstrained blasted mice, even in the lethal cases. However, no data are given to support the conclusion.

It has been suggested by several observers that lung edema is produced by injury to the hypothalamus. No data are given to support this conclusion.

A similar series of experiments(6) were carried out by the same authors, where the mice were not rigidly constrained. They compared the lung damage (hemorrhage) in radiated (600 r, 250 v. x-ray) and unirradiated mice. They discovered that the amount of lung damage was the same for both the irradiated and unirradiated mice; but that the irradiated mice were killed more easily than the unirradiated mice.

* 5 dead out of 22

** C.S. White claims that this is not true.

In the case of animals which are exposed to blast and die quickly, death in general is not due to lung edema, but rather to extensive hemorrhage and rupture. It is suspected that if they could be made to survive these effects, they would eventually die of lung edema. In any event, it appears that injury to the central nervous system plays a role in blast injury to animals.

According to the report(7), a peak overpressure of 35 psi is required to produce serious injury to persons exposed to blast. This number is consistent with an estimate by C.S. White(1) based on the data presented below.

Table 2.1

Overpressure in PSI for Indicated Mortality

Animal Species	1% Incident	1% Reflected	50% Incident	50% Reflected	99% Incident	99% Reflected
Mouse	7	20	11	30	15	44
Rabbit	9	25	12	33	15	44
Guinea Pig	10	28	13	37	17	48
Rat	10	25	14	39	18	53
Man*	20	35	40	50	55	65

* Estimate based on animal data.

It should be noted that in the above data the animal was subjected to both the incident and the reflected wave. Since the reflected overpressure exceeds the incident overpressure it is clear that the peak overpressure in the above cases is the reflected overpressure.

Dr. Cassen, of UCLA, also agrees that 35 psi is about the correct lethal overpressure for unconstrained animals. He has some disagreement with Dr. White on the effect of head shielding. This disagreement is not significant as far as our interests go, however.

We conclude that a peak overpressure of 35 psi is required to produce serious to fatal direct blast injuries to exposed personnel and that all such injured personnel will be incapacitated for a period of about 24 hours. Further, all personnel who survive direct blast injuries for a period of one hour will survive.

APPENDIX A

THE VARIATION OF $u(t)$ AND $\rho(t)$ BEHIND THE SHOCK FRONT

THE VARIATION OF u(t) AND $\rho(t)$ BEHIND THE SHOCK FRONT

These calculations are made under the following assumptions:

1. The flow behind the shock front is adiabatic and isentropic

2. The shock variables are a function of distance behind the shock front in a coordinate system moving with the shock front.

Both of these assumptions are sufficiently accurate for our purposes. It is clear, of course, that the shock intensity decreases as the shock moves outwards and that (2) is not strictly correct. This variation is slow, however, compared to the rate of decrease of the shock parameters of interest,

p_s = stagnation pressure behind shock

ρ_s = stagnation density behind shock

C_s = stagnation sonic speed behind shock

v = particle speed behind shock

M = Mach number behind shock, with $M = M_1$ initially (just behind the shock).

In a coordinate system fixed with respect to the ground let

u(t) = particle speed behind shock

U_o = shock propagation speed

$M_o = U_o/C_o$ shock propagation Mach number, where C_o is ambient sonic speed.

In either coordinate system $p = p(-x^1/U_o) = p(t)$ is the pressure behind shock where x^1 = distance from the shock front (negative measured from the shock front) in the coordinate system fixed with respect to the front and t is the time after passage of the shock front. Similarly $\rho = \rho(-x^1/U_o) = \rho(t)$ is the density behind shock and

$$v(-x^1) = U_o - u(t) = U_o - u(-x^1/U_o)$$

$$C(-x^1) = \text{sonic speed behind the shock.}$$

Now, by definition of M^2

$$M^2 = \frac{v^2}{C^2} = \frac{v^2}{C_s^2}\left(1 + \frac{\gamma-1}{2} M^2\right)$$

or

$$(1) \qquad M^2 = \frac{v^2/C_s^2}{1 - \frac{\gamma-1}{2}\frac{v^2}{C_s^2}}$$

Using the adiabatic law and assuming isentropic behavior, one has

$$(2) \qquad p_s = p\left(1 + \frac{\gamma-1}{2} M^2\right)^{\gamma/\gamma-1}$$

and

$$(3) \qquad \rho_s = \rho\left(1 + \frac{\gamma-1}{2} M^2\right)^{1/\gamma-1}$$

Using (1) we find

$$(4) \qquad 1 + \frac{\gamma-1}{2} M^2 = \frac{1}{1 - \frac{\gamma-1}{2}\frac{v^2}{C_s^2}} = \frac{1}{1 - \frac{\gamma-1}{2C_s^2}\left[U_o - u(t)\right]^2}$$

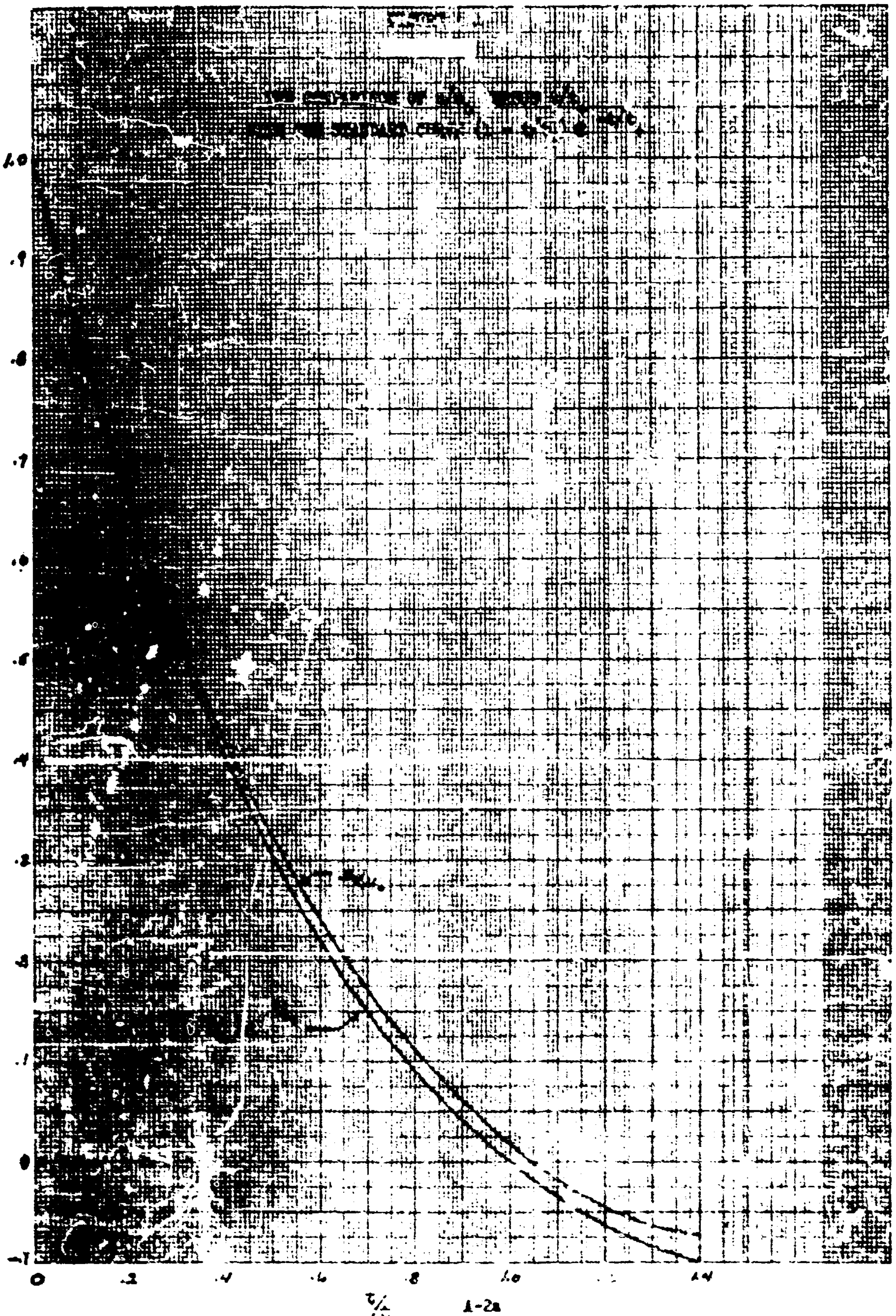
Equation (4) gives us the basic relation between M in the moving system and u(t) in the ground system.

Now, the best experimental field data on shock properties are the data on overpressure versus time. We shall confine the discussion to clean shocks and use the empirical overpressure-time curve, determined by field measurements, to derive the variation of ρ and u behind shock. Thus, one has empirically

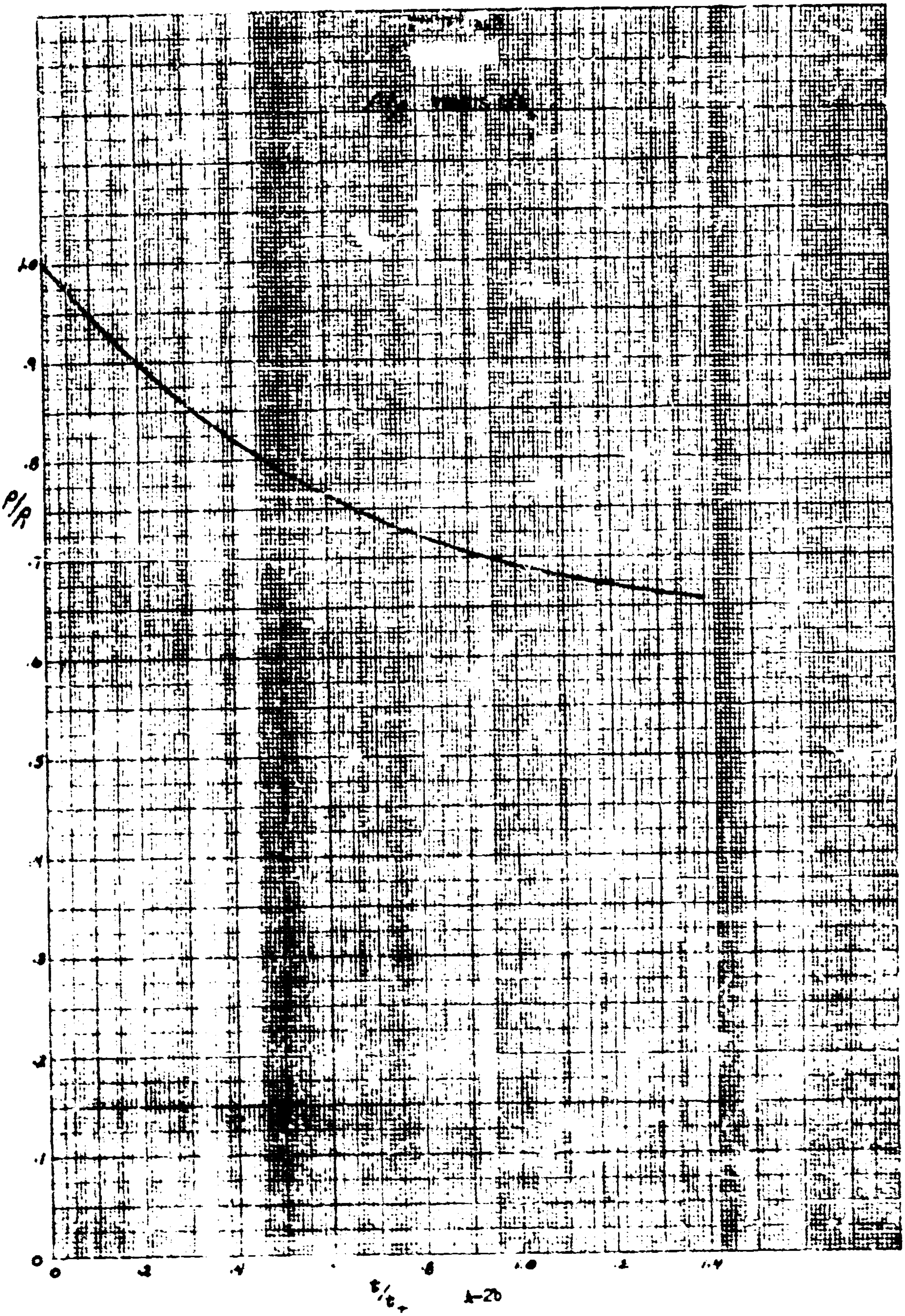
$$(5) \qquad p_{ov} = p - p_o = (p_1 - p_o)\, e^{-t/t_+}\left(1 - t/t_+\right)$$

where p_1 is the static pressure just behind shock and t_+ is the positive phase duration.

III



1-2a


1.0
.9
.8
.7
.6
.5
.4
.3
.2
.1
0
P/R
0
.2
.4
.6
.8
1.0
1.2
1.4
t/t_r

Using (2),

$$p_{ov} = p - p_0 = \frac{p_1\left(1+\frac{\gamma-1}{2}M_1^2\right)^{\gamma/\gamma-1}}{\left(1+\frac{\gamma-1}{2}M^2\right)^{\gamma/\gamma-1}} - p_0 \tag{6}$$

Substituting for $1 + \gamma\text{-}1/2\, M^2$ from (4) and equating (5) and (6) we obtain for u

$$u(t) = u_0 + \sqrt{\frac{2}{\gamma-1}}\, C_s\left[1-\left(\frac{p_1}{p_{s1}}\right)^{\gamma-1/\gamma}\right]^{1/2} - \sqrt{\frac{2}{\gamma-1}}\, C_s\left[1-\left\{\frac{p_0+(p_1-p_0)(1-t/t_+)\epsilon^{-t/t_+}}{p_{s1}}\right\}\right. \tag{7}$$

where

$$p_{s1} = p_1\left[1+\frac{\gamma-1}{2}M_1^2\right]^{\gamma/\gamma-1}$$

is the stagnation pressure just behind shock and

$$u_0 = U_0 - \sqrt{\frac{2}{\gamma-1}}\, C_s\left[1-\left(\frac{p_1}{p_{s1}}\right)^{\gamma-1/\gamma}\right]^{1/2}$$

is the maximum particle velocity in the ground system.

Similarly from (3)

$$\rho(t) = \rho_1\left\{\frac{p_0+(p_1-p_0)\epsilon^{-t/t_+}(1-t/t_+)}{p_1}\right\}^{1/\gamma} \tag{8}$$

where ρ_1 is the maximum density behind shock. Thus, (7) and (8) give us the observed variation of u(t) and $\rho(t)$ as a function of the measured overpressure variation.

Figures A.1 and A.2 show u/u_0 and ρ/ρ_1 plotted vs. t/t_+. For comparison purposes, the function $(1 - t/t_+)\,\epsilon^{-t/t_+}$ is shown plotted on Figure A.1. It is clear that, to a good approximation, u/u_0 declines as $(1 - t/t_+)\,\epsilon^{-t/t_+}$. For convenience of the reader, the computational values of ρ/ρ_1, u/u_0 and $(1 - t/t_+)\,\epsilon^{-t/t_+}$ are given in Table A.1 for a shock of 10 psi overpressure. One notes the slightly longer positive duration of the wind speed u compared to the duration of the overpressure.

One should also note that if ρ_0 is the ambient density, then

$$\frac{\rho_1}{\rho_0} = \frac{(\gamma+1)M_0^2}{(\gamma-1)M_0^2+2}$$

where

$$M_0 = U_0/C_0 = \text{Mach number of shock front.}$$

Table A.1

Values of u/u_0 And ρ/ρ_1 Computed on the Assumption
That The Overpressure Follows the Law
$p_{ov} = p_1(1 - t/t_+)\,\epsilon^{-t/t_+}$ (10 psi shock)

t/t_+	$(1 - t/t_+)\,\epsilon^{-t/t_+}$	u/u_0	ρ/ρ_1	ρ/ρ_0
0	1.000	1.000	1.000	1.446
.2	.655	.561	.898	1.30
.4	.402	.415	.820	1.19
.6	.220	.244	.763	1.10
.8	.090	.113	.720	1.04
1.0	.000	.022	.691	1.00
1.2	- .060	- .045	.670	.97
1.4	- .099	- .072	.657	.95

Further, one should note that if an average value of ρ/ρ_0 is used in the calculation, very little error will be made. Thus in the example above, one would set $\bar{\rho}/\rho_0 = 1.22$ and use $\bar{\rho}$ instead of ρ_0. Thus, in our translation calculations we have used $\bar{\rho}$ to simplify the procedure. The error in so doing is not significant.

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IV. COMBINED INJURIES

In this Chapter we consider the combined effect on personnel of nuclear radiation, thermal radiation and blast from the detonation of a nuclear weapon. Since this report is concerned basically with casualties from an operational point of view rather than from a medical point of view, our discussion will be appropriately limited. Specifically we shall consider only those situations for which no single type injury is lethal or seriously incapacitating, but where a combination of two or more may be so. We shall show that no class of combined injury is important except thermal-nuclear injury. It should be noted that considerations here are limited to typical situations. No doubt certain bizarre environmental conditions may arise for which other classes of combined injury may be important. However, it is felt that the occurrence of such situations will be relatively unusual so that they may reasonably be neglected here.

We shall first show that combined injuries which involve blast injury may be neglected. Now, blast injury is devided into two categories: direct blast injury and indirect blast injury. For the case of direct blast injury, that is, injury which results from a sharp increase of the static pressure, we find from Chapter III that all personnel who receive serious injury are incapacitated for about 24 hours. Less serious injuries, such as ear drum rupture, do not appear to cause incapacitation. Further, all deaths which occur will probably occur within one hour and all injured survivors will recover to normal within the 24 hour period of incapacitation. There is, no doubt, a range of direct blast injury for which the above general statements do not apply; however, all the data indicates that the greater majority of cases will fall outside this range. Thus, combined injuries which involve direct blast injury fall in two categories, those injuries for which the injury alone is either lethal or seriously incapacitating and those for which the injury is relatively minor. Accordingly, combined injuries which involve direct blast injury are not important from an operational point of view. In addition, personnel who are exposed both to the thermal field and to the direct blast field receive either an overwhelming thermal dose or an overwhelming γ-ray dose at blast levels which could cause direct injury (35 psi). We draw a similar conclusion for indirect blast injury, that is, injury which results from flying debris and from personnel being thrown to the ground or against rigid objects. As indicated in Chapter II, flying debris resulting from the wind behind the shock front is not an important casualty producing agent. Thus, we need to consider only those injuries which result from personnel being thrown against the ground, etc. The only injuries brought about in this manner which are not considered to be minor are bone fracture and concussion. Injuries of this type are at best seriously incapacitating, particularly when more or less immediate medical attention is not available. It, therefore, follows that combined injury involving indirect blast injury need not be considered.

Combined thermal radiation injury and nuclear radiation injury in accordance with the preceding paragraph is the only class of combined injury we have left to consider. Whether or not this class of combined injury will assume a significant role will depend greatly on the particular situation. That is, the effect of shielding, clothing and evasive tactics on the extent of thermal injury is quite pronounced. In the case of completely exposed personnel in summer clothing who employ no evasive tactics, combined thermal-nuclear injury will not play a significant role for weapon sizes in excess of about 10 KT. This is clear from Table 1, since immediate incapacitation occurs for unprotected personnel who are exposed to 20 cal/cm^2 or more thermal energy and since 240 rems is regarded as the minimum amount of nuclear radiation required to produce any nuclear radiation injury. Further, it is clear that shielding, heavy clothing and evasive tactics will tend to make thermal-nuclear injury important at higher weapon yields. The effect of shielding, heavy clothing and evasive tactics does not properly fall within the confines of this contract and will be considered in detail under another contract.

Table 1

Weapon Yield Kilotons	Nuclear Radiation Dose REM's
2	> 1000
4	> 1000
10	443
20	< 240
100	< 240
1000	< 240
10000	< 240

In Chapter II of this report, the effect on personnel of the thermal radiation from nuclear explosions is discussed in detail. It is concluded that 3° whole-body burns will be relatively rare and that such burns will be immediately incapacitating in disaster situations since prompt medical attention will be unavailable. In the case of 2° whole-body burns it is shown that if the face, legs or the back of the hands are extensively involved, then total incapacitation will occur within about 3 hours. Further, serious incapacitation will occur more or less immediately. Accordingly, combined thermal-nuclear injury is important only for those situations where the thermal injury does not involve the critical areas mentioned. Early incapacitation from 2° whole-body burns is due to hypovolemic shock and will occur according to Table 2 below.

Table 2

Time to Hypovolemic Shock versus
Percent Body Area Burned for Median 2° Burns

Body Area Percent	Time to Shock Hours
10	(53)*
15	25.8
20	17.2
25	13.1
30	10.7
40	7.6
50	6.2

* Shock does not occur.

Now, the data on combined thermal and nuclear injury is scant. Figures 1, 2, 3 and 4, however, show the general features of the problem. The data are taken on rats with an LD-50 of about 700 r. One notes that there is little increase in deaths versus dosage until the 500 r level is reached. If the human LD-50 is taken at 550 r, therefore, the human level corresponding to 500 r in the rat would be about 400 r. Thus, the serious affects of combined thermal-nuclear injury appear only at relatively high nuclear dosage.

Aside from the effect of the combined injury on early fatalities, which is shown clearly in the Figures, very little can be said quantitatively about cases of non-lethal combined thermal-nuclear injury. This much can, however, be said.

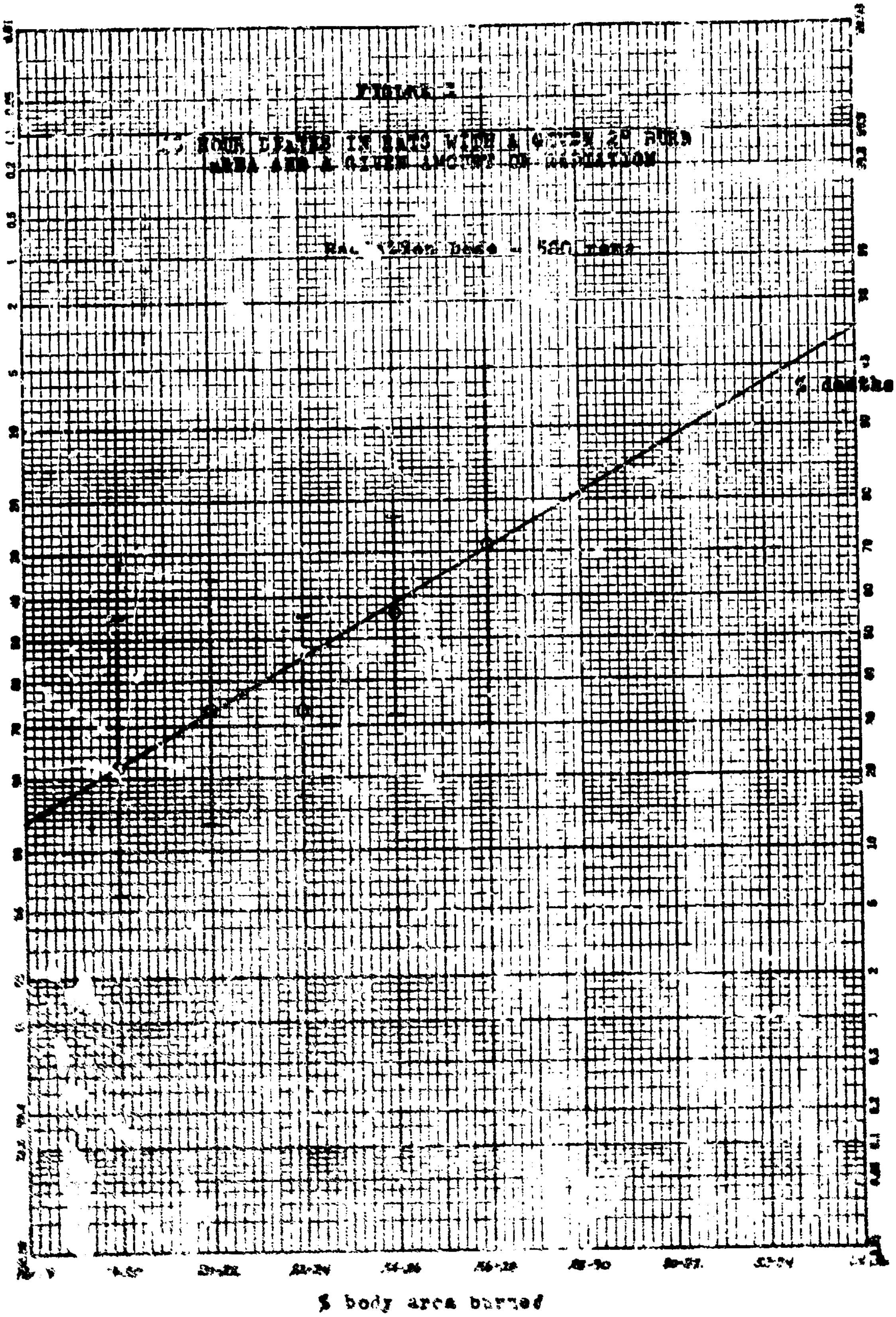
1. Referring to Table 2, the 15% to 20% whole-body burn combined with 400 r of radiation would undoubtedly be fatal to the human in 48 hours unless intra-

veneous fluid therapy were available; and this is not likely to be available under disaster conditions. 400 r is enough to cause nausea and to prevent oral fluid therapy.

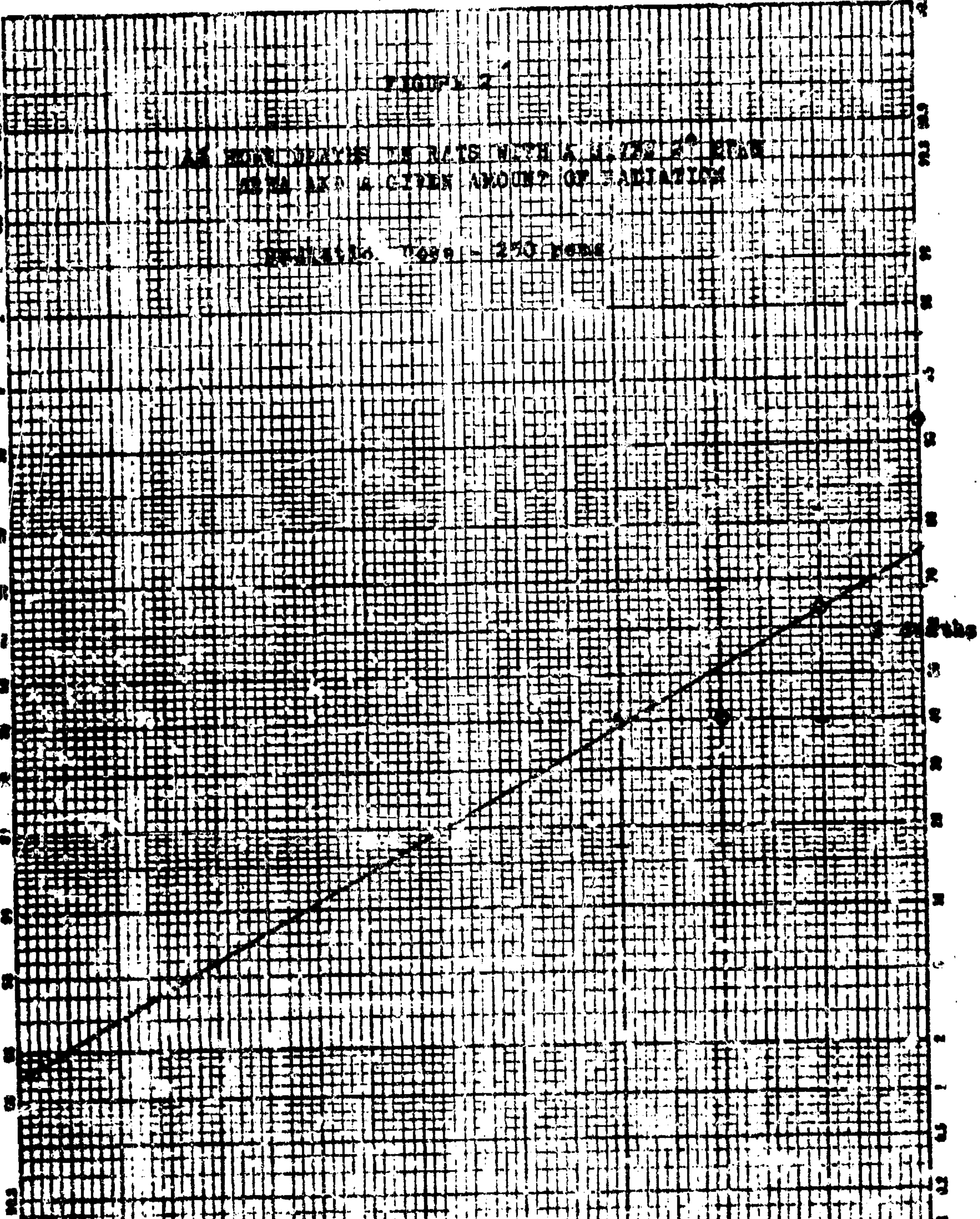
2. A radiation dose will increase the susceptability to infection for smaller area burns.

3. Nausea with consequent oral fluid loss will decrease the time to shock.

In summary, the data on combined thermal-nuclear injury are sparse. However, before the day of the use of intravenous fluid therapy in treatment of human burn cases about 1/2 of the burn cases survived a 33% second degree area burn. One notes that this is also true of the rats. The humans who went into shock, however, undoubtedly had specific treatment for shock symptoms. In any event, the rat data are presumed to apply approximately to humans.



% body area burned



% body area burned

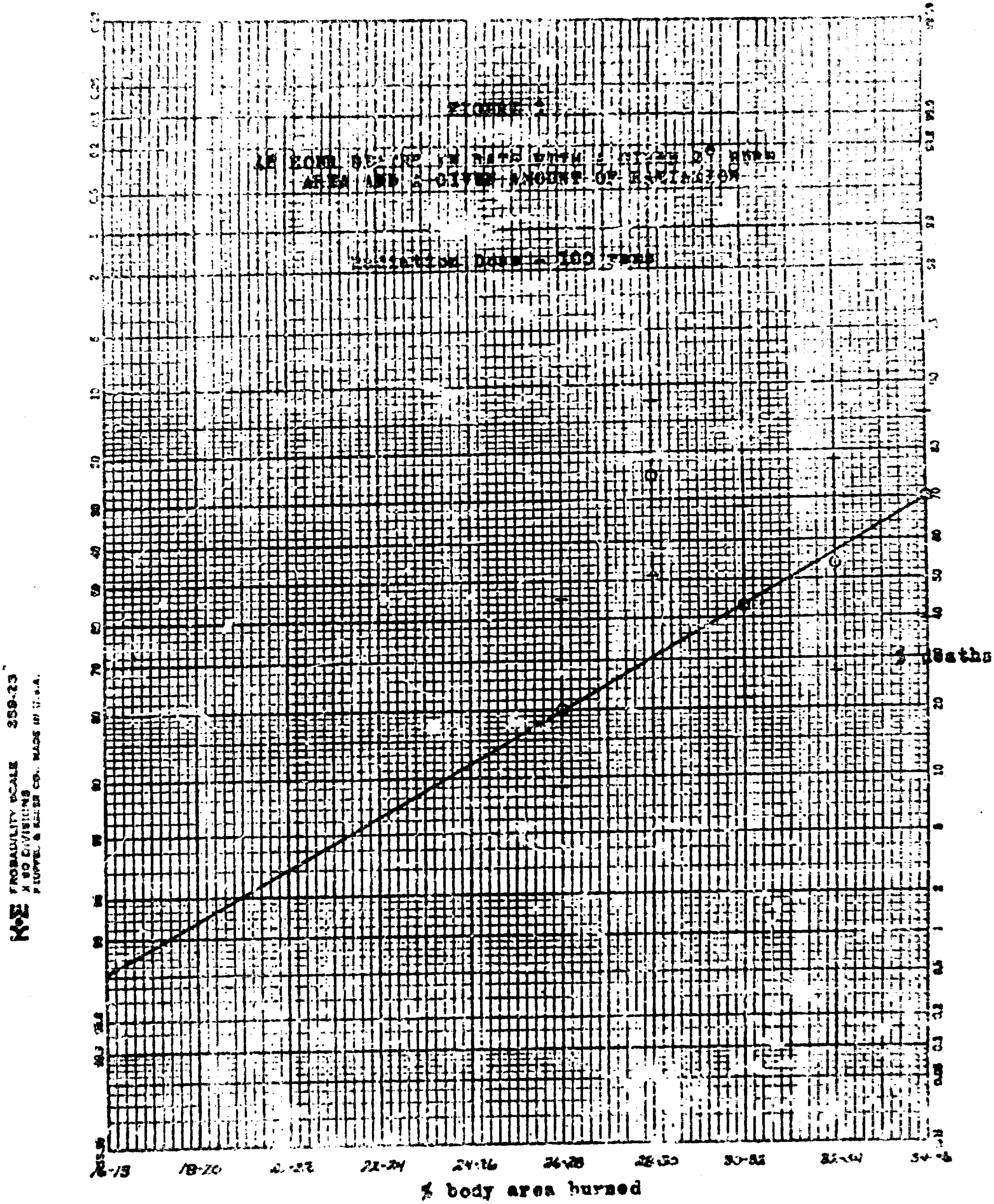
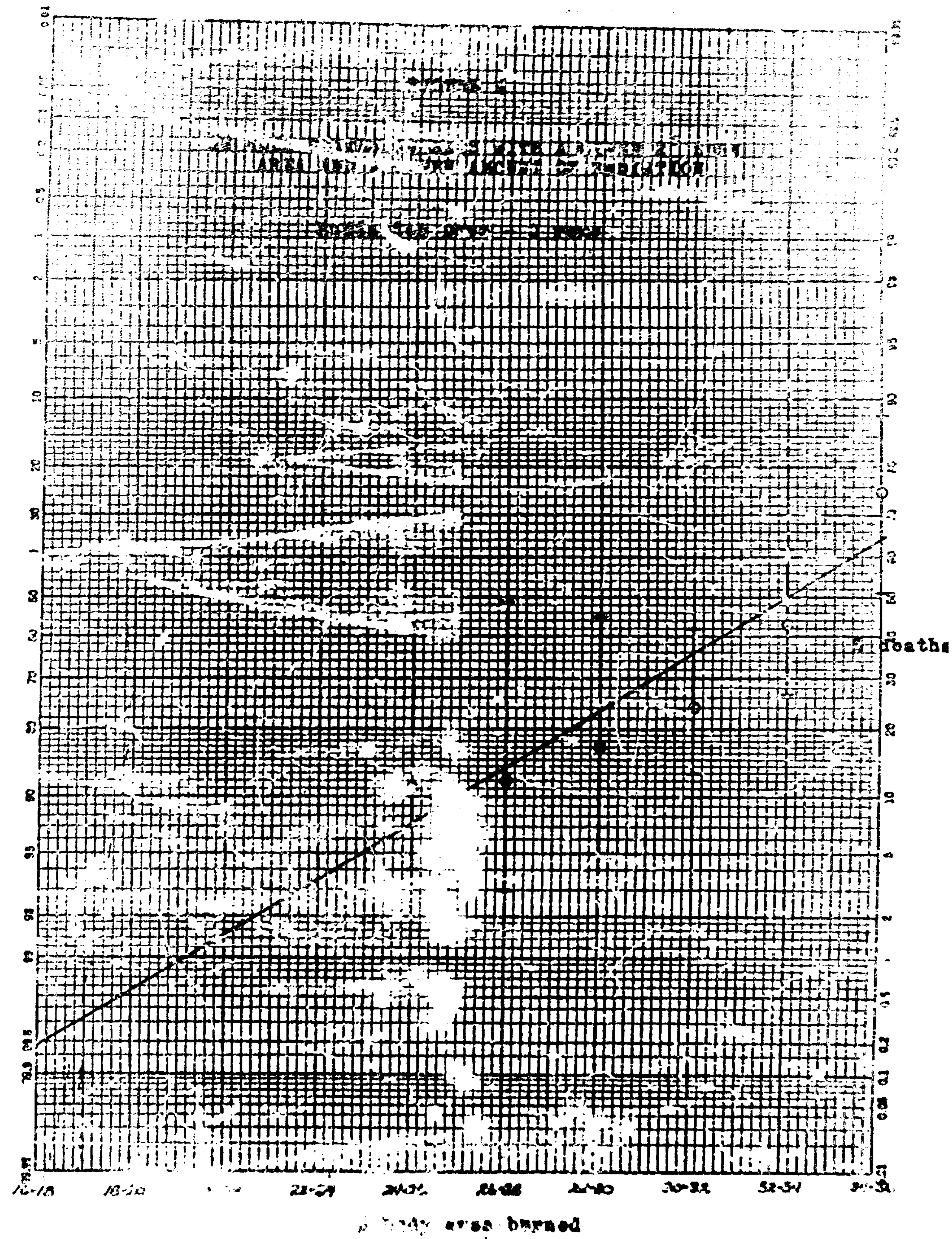


FIGURE
% deaths
18-20
22-24
24-26
26-28
30-32
% body area burned



deaths
body area burned